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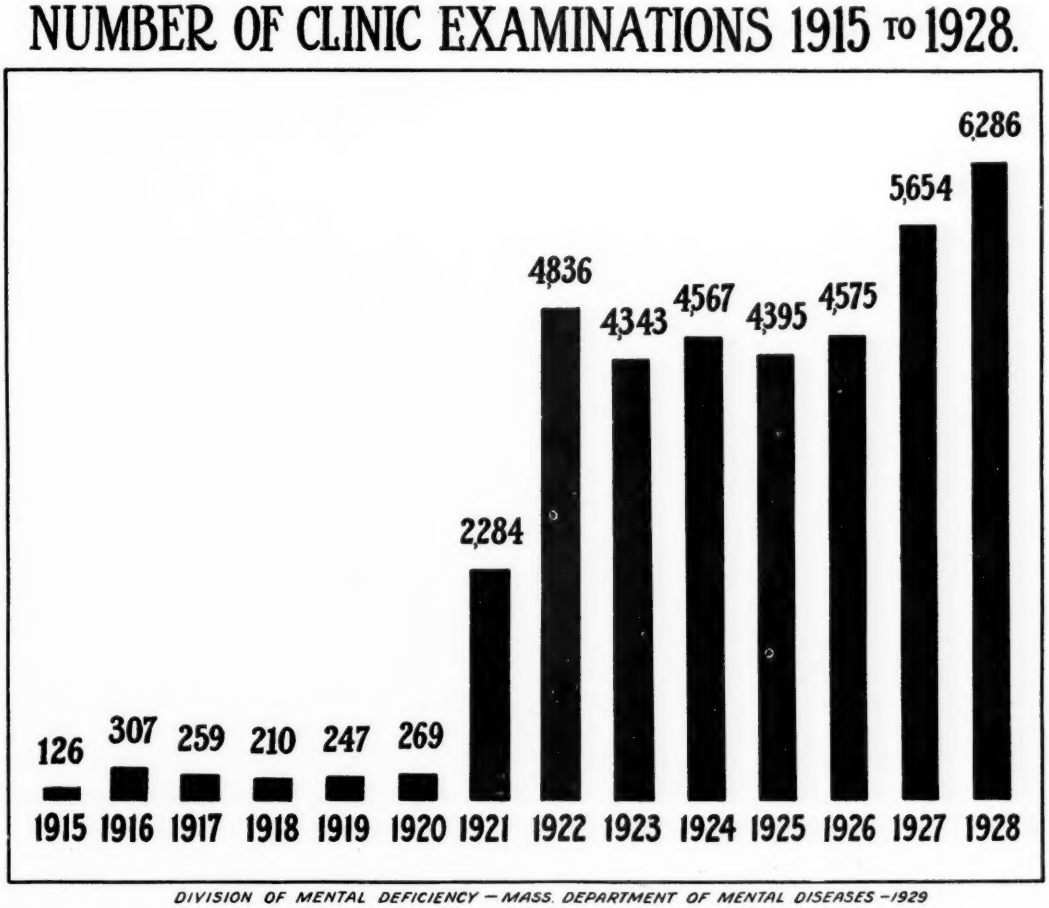
# THE RELATIONSHIP BETWEEN PHYSICAL DEFECTS AND INTELLIGENCE*

BY NEIL A. DAYTON, M.D.†

THE material for the present paper was taken from the physical examinations of 14,379 retarded children in the public schools of Massachusetts. The physical examination comprises one section of the Fernald Ten Point Scale examination which covers ten different fields, and makes inquiry into the physical, pedagogic, psychologic, social and personal histories of each retarded child examined. The examinations were made during the period 1921-27 by the traveling school clinics of the Walter E. Fernald State School, the Wrentham State School, the Boston Psychopathic Hospital, and the Foxborough State Hospital.

GRAPH I




The traveling school clinic idea provides a very complete psychiatric examination for all backward school children. It was devised and placed in operation by the late Dr. Walter E. Fernald who sent out the first traveling clinic from the Waverley School in 1915. Later, in 1917, Dr. George L. Wallace sent out the second traveling clinic from the Wrentham State School. As time went on, however, it was soon evident that these two clinics would not be able to examine all the backward children throughout the entire state, and the formation of addi-

*Read at the annual meeting of the American Association for the Study of the Feebleminded, Atlanta, Georgia. May 13, 14, 15, and 16, 1929.

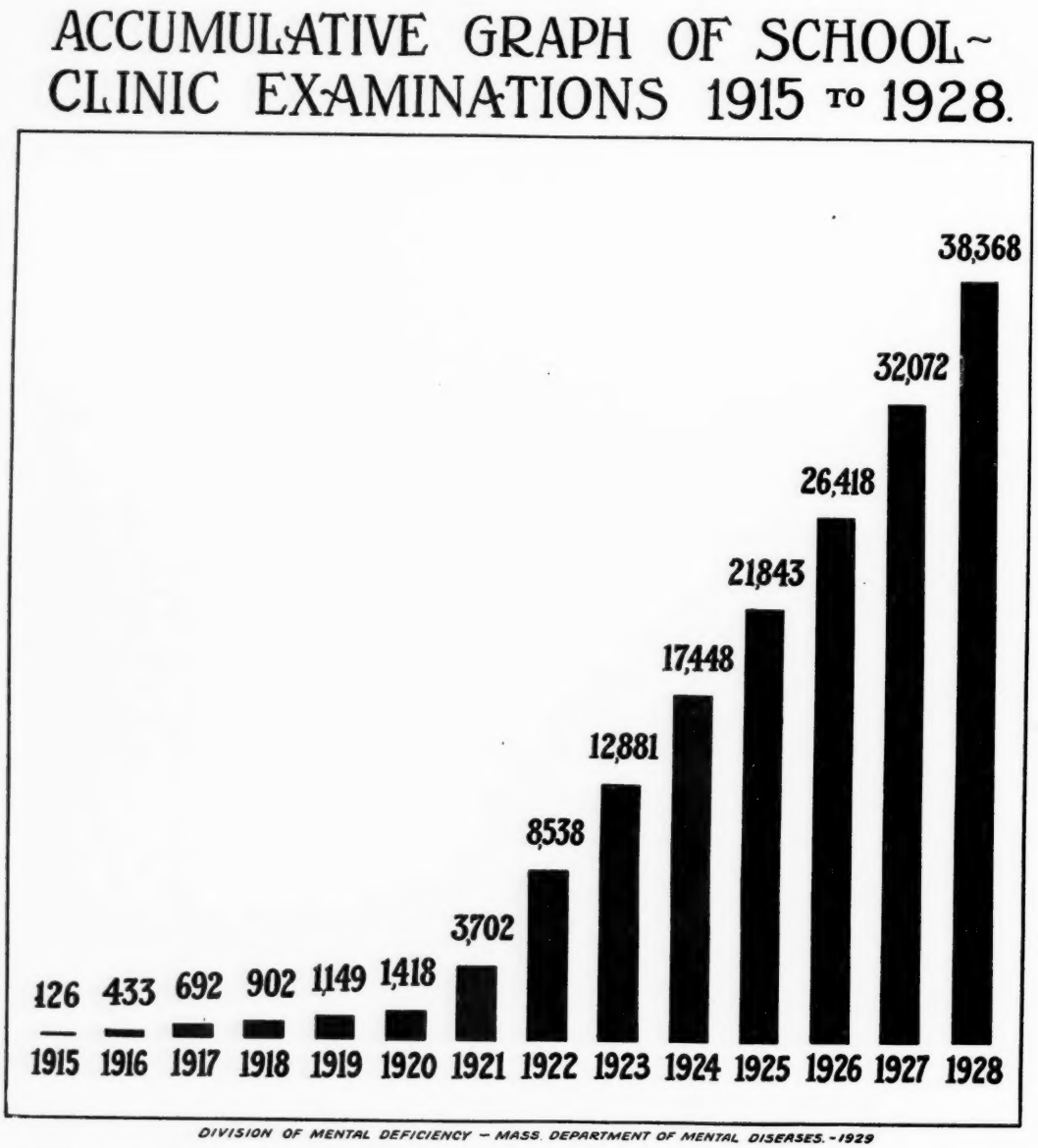
From the Division of Research, Massachusetts Department of Mental Diseases, Research Project No. I, Article IV. The writer was aided in the preparation of this paper by Dorothy G. Gooch, A.B., and Laurette Bogott, A.B., Assistants in Research.

†For record and address of author see "This Week's Issue," page 282

tional units became imperative. Dr. Fernald placed the matter before the Commissioner of Mental Diseases, Dr. George M. Kline, and in 1921, as a result of their collaboration, new clinics were created to operate from each of the fourteen institutions under the Department of Mental Diseases. Thus, for the first time an adequate state-wide system of examination was made possible.

The growth in the number of examinations completed each year is outlined in Graph I. This shows the humble beginning in 1915, when 126 children were examined, and the steady increase up to 1928 when 6,286 were examined. The striking increase in 1921 is due, of course, to the simultaneous operation of fourteen clinics. The fifteenth clinic was started in January 1928, by Dr. George E. McPherson,

GRAPH II




Dr. Kline saw that the withdrawal of a psychiatrist from the medical staffs of the various hospitals was impracticable, and therefore increased the quota of each institution by one physician and one psychologist to carry on this important work. Dr. Payson Smith, Commissioner of Education, took an active part in framing the law, relating to retarded children and in outlining and enforcing the school clinic regulations which have contributed so materially to the school clinic system.

Superintendent of the Belchertown State School.

Graph II outlines the accumulation of examinations. It shows that a total of over 38,000 examinations have been conducted by the clinics during the thirteen years in which they have been in operation.

## PURPOSES

The 14,379 first examinations[1] considered in this study have been used in an attempt to determine whether or not there is any relationship

between weight variations and the degree of the mental defect observed. An effort was also made to determine whether or not any relationship existed between the presence or absence of physical defect and the intelligence.

1. These examinations were made from 1921 to 1927 by Dr. Esther S. B. Woodward of the W. E. Fernald State School, Dr. Alice M. Patterson of Wrentham State School, Dr. Cornelia B. J. Schorer of Foxborough State Hospital, and Drs. Marianna Taylor, Margareta A. Ribble, and Julia Deming of Boston Psychopathic Hospital. The co-öperation of Drs. Ransom A. Greene, George L. Wallace, C. Macfie Campbell and Roderick B. Dexter has been greatly appreciated.

Approximately 16,500 examinations have been studied. 14,379 are first and the remainder re-examinations. Only first examinations were used to avoid duplication.

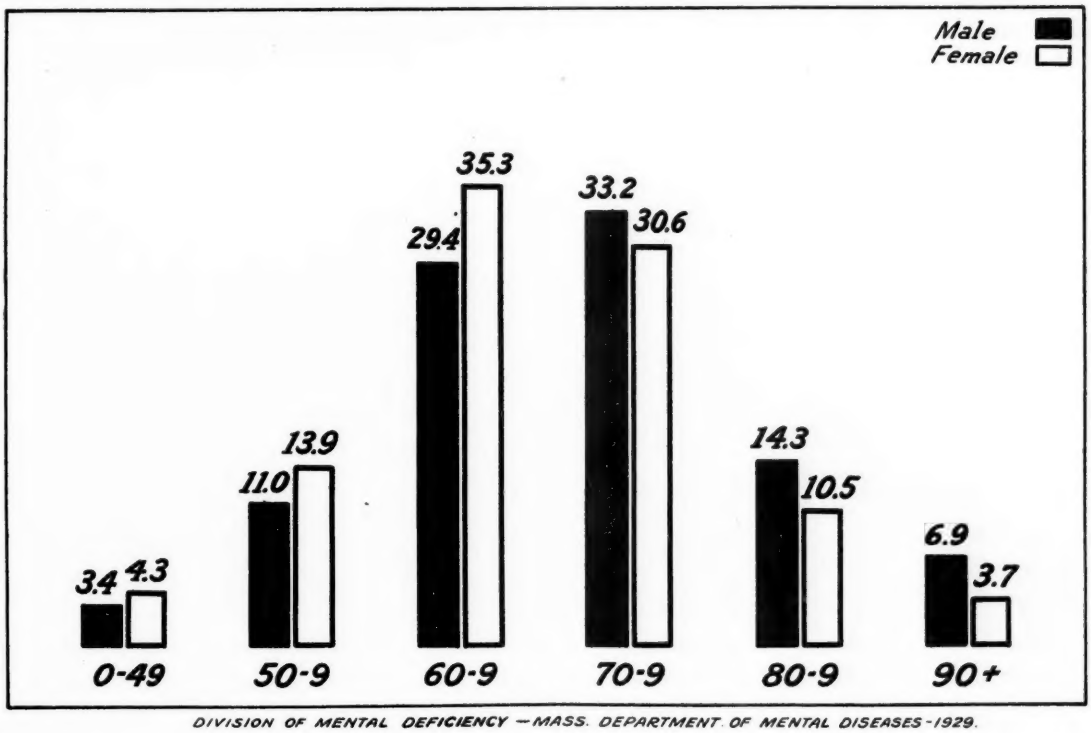
### INTELLIGENCE

Graph III and Table A show the distribution of intelligence quotients among these retarded school children. It will be noted that there are twice as many boys as girls, and that the mean intelligence of the boys is 2.6 points higher than that of the girls.[2] The Stanford revision of the Binet-Simon was used for the determination of mental age in all examinations.

2. At this point we have dropped 132 males and 72 females whose intelligence quotients are unknown, as both numbers are less than 2% of the totals. The new total will be 14,175, 9,138 males and 5,037 females.

GRAPH III

**PERCENTAGE DISTRIBUTION OF INTELLIGENCE QUOTIENTS OF 14,379 RETARDED PUBLIC SCHOOL CHILDREN, BY SEX.**




DIVISION OF MENTAL DEFICIENCY — MASS. DEPARTMENT OF MENTAL DISEASES - 1929.

TABLE A

DISTRIBUTION OF INTELLIGENCE QUOTIENTS OF 14,379 RETARDED PUBLIC SCHOOL CHILDREN, BY SEX

| I. Q. Groups | Total | 0-.49 | .50-.59 | .60-.69 | .70-.79 | .80-.89 | .90-.99 | 1.00-1.09 | 1.10-1.19 | 1.20+ | Unk. |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Male | 9270 | 322 | 1023 | 2733 | 3084 | 1330 | 460 | 122 | 33 | 31 | 132 |
| Female | 5109 | 223 | 712 | 1807 | 1568 | 538 | 139 | 38 | 10 | 2 | 72 |
| Total | 14379 | 545 | 1735 | 4540 | 4652 | 1868 | 599 | 160 | 43 | 33 | 204 |

| | Mode | Median | Mean | S. D. |
|---|---|---|---|---|
| Male | .70-.79 | 71.5 | 71.6 | ± .12 |
| Female | .60-.69 | 68.7 | 69.0 | ± .16 |
| Total | .70-.79 | 70.5 | 70.7 | ± .10 |

### WEIGHT VARIATIONS AND INTELLIGENCE

The relationship between the weight factor and intelligence will be considered first. We

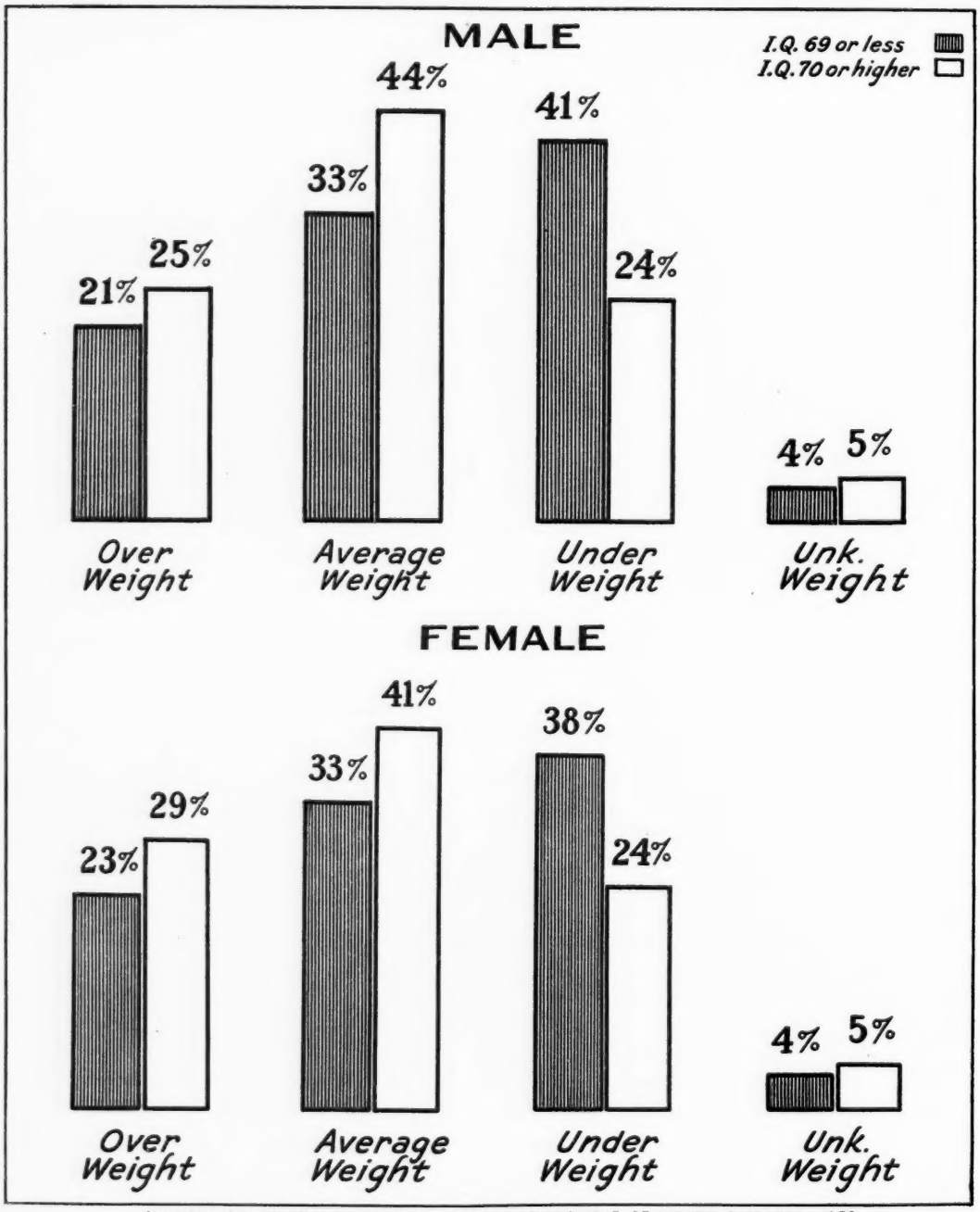
TABLE B

DISTRIBUTION OF 14,175 RETARDED SCHOOL CHILDREN INTO WEIGHT GROUPS, BY SEX

| | Total | Over-Average Weight | | Average Weight | | Under-Average Weight | | Unknown Weight | |
|---|---|---|---|---|---|---|---|---|---|
| | | Number | Per Cent. | Number | Per Cent. | Number | Per Cent. | Number | Per Cent. |
| Male | 9,138 | 2,131 | 23.3 | 3,612 | 39.5 | 2,915 | 31.9 | 480 | 5.2 |
| Female | 5,037 | 1,326 | 26.3 | 1,863 | 37.0 | 1,596 | 31.7 | 253 | 5.0 |
| Total | 14,175 | 3,457 | 24.4 | 5,475 | 38.6 | 4,511 | 31.8 | 733 | 5.1 |

GRAPH IV

## PERCENTAGE DISTRIBUTION OF OVER-AVERAGE, AVERAGE AND UNDER-AVERAGE WEIGHT GROUPS BY INTELLIGENCE QUOTIENTS AND SEX.



have divided the children into three groups, over-average weight, average weight, and under-average weight.[3] The results of this division are shown in Table B.

3. These groups are based on the Baldwin-Wood Tables for average children. We have taken the weight based on height, age and sex, and have made an arbitrary allowance of ten per cent. above and below the mean weight to constitute the average group. Weights above or below the limits of the average group are, therefore, above or below average weight.

At once there arises the question of a control group for comparison. Our figures in themselves have been subjected to a certain degree of control in the matter of comparison with the Baldwin-Wood Tables, which are based on averages.

TABLE C

DISTRIBUTION OF GROUPS OVER-AVERAGE, AVERAGE, AND UNDER-AVERAGE WEIGHT, BY SEX, INTO INTELLIGENCE GROUPS

(1) I. Q. .69 or Lower
(2) I. Q. .70 or Higher

| Sex | Total | I. Q. .69 or lower | I. Q. .70 or higher |
|---|---|---|---|
| | *Over-Average Weight* | | |
| Male | 2131 | 861 | 1270 |
| Female | 1326 | 649 | 677 |
| Total | 3457 | 1510 | 1947 |
| | *Average Weight* | | |
| Male | 3612 | 1357 | 2255 |
| Female | 1863 | 922 | 941 |
| Total | 5475 | 2279 | 3196 |
| | *Under-Average Weight* | | |
| Male | 2915 | 1673 | 1242 |
| Female | 1596 | 1044 | 552 |
| Total | 4511 | 2717 | 1794 |
| | *Unknown Weight* | | |
| Male | 480 | 187 | 293 |
| Female | 252 | 127 | 125 |
| Total | 732 | 314 | 418 |
| | *Total of All Weight Groups* | | |
| Male | 9138 | 4078 | 5060 |
| Female | 5037 | 2742 | 2295 |
| Total | 14175 | 6820 | 7355 |

At the same time, it was thought best to divide our own group with particular reference to intelligence in an effort to establish a control within our sample. Consequently, in the examinations, weight groups were divided into the following intelligence groupings:

I. Intelligence quotient .69 or lower.
II. Intelligence quotient .70 or higher.

This sharply separates our so-called feebleminded from those not to be considered as feebleminded.

The well-known fact that intelligence is a continuous variable will probably bring forth a protest at our establishment of a sharp line of demarcation at .70. However, the difficulty of establishing a dividing line for mental deficiency is only too evident to the writer, and this limit is simply accepted as a convenience, without any great belief in its infallibility.

The results of this division of weight groups by intelligence are shown in Graph IV and Table C. The figures outline for us quite definitely the fact that under-weight is consistently associated with mental defect in both sex groups. The distribution for our groups above .70 presents the normal bell-shaped curve, that is, the average group exceeds the under-weight and over-weight groups by comfortable margins. When we consider the feebleminded group, however, our curve is markedly skewed. It would seem, then, that the factor of under-weights is not of great importance in children who are not feebleminded. However, the children considered as feebleminded seem to have a very definite tendency to belong in the under-weight groups.

### AVERAGE INTELLIGENCE OF GROUPS OVER-AVERAGE, AVERAGE, AND UNDER-AVERAGE IN WEIGHT

From the foregoing, we see that there is an apparent association between the lower degrees of mental defect and the physical factor of under-weight. To develop this point more clearly, however, we calculated the mean intelligence quotients of the three weight groups by sex, as shown in Graph V and Table D. The mean intelligence of groups over-average in weight was compared with those average in weight. Here we found that there was practically no difference whatsoever. However, when we compared the groups average in weight with those under-average in weight, we found that the intelligence showed a difference of 5.4 points of the I. Q. scale for the males and 4.3 points for the females. It is noted that in general under-weight has less effect on the I. Q. distribution of the females than on that of the males. There is a much higher percentage of low I. Q.'s in the under-weight group of males than in the under-weight group of females. The standard deviation of the mean intelligence quotients of the above mentioned weight groups was computed. As the difference between the mean intelligence of groups average and under-average in weight are 18 and 11 times the standard deviation of the difference, we cannot question the significance of our findings. In table D it is of interest to note that the medians do not differ greatly from the means. From the results which we have obtained we can say quite definitely that from the material of this sample there is a definite association between the physical factor of under-weight and lower intelligence in retarded school children.

### PHYSICAL DEFECTS AND INTELLIGENCE

The next point that we wish to take up is the consideration of the relationship between various physical defects and intelligence. We first

GRAPH V

## DIFFERENCE BETWEEN MEAN INTELLIGENCE QUOTIENTS OF GROUPS OVER-AVERAGE, AVERAGE AND UNDER-AVERAGE IN WEIGHT.

| SEX | OVER-AVERAGE WEIGHT | | | AVERAGE WEIGHT | | | UNDER-AVERAGE WEIGHT | | | DIFFERENCE | $\sigma_d$ | DIFF. / $\sigma_d$ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | N. | MEAN I.Q. | S.D. | N. | MEAN I.Q. | S.D. | N. | MEAN I.Q. | S.D. | | | |
| M. | 2131 | 73.2 | ±.26 | 3585 | 73.3 | ±.20 | | | | -.1 | .32 | 0 |
| F. | 1326 | 70.3 | ±.32 | 1863 | 70.3 | ±.26 | | | | 0 | .41 | 0 |
| | | | | | | | | | | | | |
| M. | | | | 3585 | 73.3 | ±.20 | 2915 | 67.9 | ±.21 | 5.4 | .29 | 18.6 |
| F. | | | | 1863 | 70.3 | ±.26 | 1596 | 66.0 | ±.27 | 4.3 | .37 | 11.6 |
| | | | | | | | | | | | | |
| M. | 2131 | 73.2 | ±.26 | | | | 2915 | 67.9 | ±.21 | 5.3 | .33 | 16.0 |
| F. | 1326 | 70.3 | ±.32 | | | | 1596 | 66.0 | ±.27 | 4.3 | .41 | 10.4 |

DIVISION OF MENTAL DEFICIENCY — MASSACHUSETTS DEPARTMENT OF MENTAL DISEASES - 1929

TABLE D

DISTRIBUTION OF INTELLIGENCE QUOTIENTS OF GROUPS OVER-AVERAGE, AVERAGE, AND UNDER-AVERAGE IN WEIGHT, BY SEX

| I. Q. Groups | Total | 0-.49 | .50-.59 | .60-.69 | .70-.79 | .80-.89 | .90-.99 | 1.00+ | Mode | Median | Mean | S. D. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *Over-Average Weight* | | | | | | | | | | | | |
| Male | 2131 | 46 | 202 | 613 | 719 | 357 | 128 | 66 | 70-79 | 72.8 | 73.2 | ± .26 |
| Female | 1326 | 51 | 158 | 440 | 450 | 159 | 45 | 23 | 70-79 | 70.3 | 70.3 | ± .32 |
| Total | 3457 | 97 | 360 | 1053 | 1169 | 516 | 173 | 89 | 70-79 | 71.8 | 72.1 | ± .20 |
| *Average Weight* | | | | | | | | | | | | |
| Male | 3612 | 99 | 317 | 941 | 1325 | 645 | 216 | 69 | 70-79 | 73.3 | 73.3 | ± .20 |
| Female | 1863 | 59 | 221 | 642 | 619 | 249 | 57 | 16 | 70-79 | 70.1 | 70.3 | ± .26 |
| Total | 5475 | 158 | 538 | 1583 | 1944 | 894 | 273 | 85 | 70-79 | 72.3 | 72.2 | ± .16 |
| *Under-Average Weight* | | | | | | | | | | | | |
| Male | 2915 | 159 | 451 | 1063 | 906 | 253 | 67 | 16 | 60-69 | 67.9 | 67.9 | ± .21 |
| Female | 1596 | 96 | 302 | 646 | 429 | 102 | 18 | 3 | 60-69 | 66.1 | 66.0 | ± .27 |
| Total | 4511 | 255 | 753 | 1709 | 1335 | 355 | 85 | 19 | 60-69 | 67.3 | 67.2 | ± .16 |
| *Unknown Weight* | | | | | | | | | | | | |
| Male | 480 | 18 | 53 | 116 | 134 | 75 | 49 | 35 | 70-79 | 73.2 | 74.6 | ± .67 |
| Female | 252 | 17 | 31 | 79 | 70 | 28 | 19 | 8 | 60-69 | 69.8 | 70.6 | ± .90 |
| Total | 732 | 35 | 84 | 195 | 204 | 103 | 68 | 43 | 70-79 | 72.5 | 73.3 | ± .54 |

made a rough grouping of physical diseases or defects by sex. Insofar as we do not wish to make any refined or detailed study of the relationship, we confined these to 12 groupings. They are as follows:

FINDINGS OF PHYSICAL EXAMINATIONS RECORDED BY SYSTEMS

| Defect | Male | Female | Total |
|---|---|---|---|
| Vision and hearing (special sense) | 2242 | 1458 | 3700 |
| Circulatory | 907 | 638 | 1545 |
| Skeletal | 819 | 385 | 1204 |
| Neurological | 685 | 373 | 1058 |
| Endocrine | 119 | 360 | 479 |
| Respiratory | 71 | 31 | 102 |
| Genito-Urinary | 26 | 22 | 48 |
| Tonsils, Adenoids and Teeth (Gastro-Intestinal) | 5638 | 3138 | 8776 |
| Stigmata | 2862 | 1477 | 4339 |
| Others | 1875 | 1220 | 3095 |
| Incomplete | 111 | 46 | 157 |
| Negative | 1298 | 601 | 1899 |
| Total Examinations | 9138 | 5037 | 14,175 |
| Total Defects | 15,244 | 9102 | 24,346 |

These groupings vary somewhat from the usual set-up, but had to be adapted to the particular problem of mental defect. For example, in no other physical disturbance would the matter of stigmata be of such importance. In mental deficiency, however, the frequency of the

phenomena renders it important that they be recorded. In like manner it may be said that practically 99% of the gastro-intestinal defects[4] are defects of tonsils, adenoids or teeth. It might be well to say that in dealing with all of these defects, all physical findings designated as "slight" or "somewhat" were eliminated. As we wanted our findings to be clear-cut we have not taken doubtful recordings.

| | | | |
|---|---|---|---|
| Genito-Urinary | 26 | 10 | 16 |
| Incomplete | 111 | 25 | 86 |
| Negative | 1298 | 396 | 902 |
| Total Defects | 15244 | 7941 | 7303 |
| No. of Examinations | 9138 | 4078 | 5060 |
| Defect per child* | 1.6 | 1.9 | 1.4 |

*Incomplete examinations excluded.

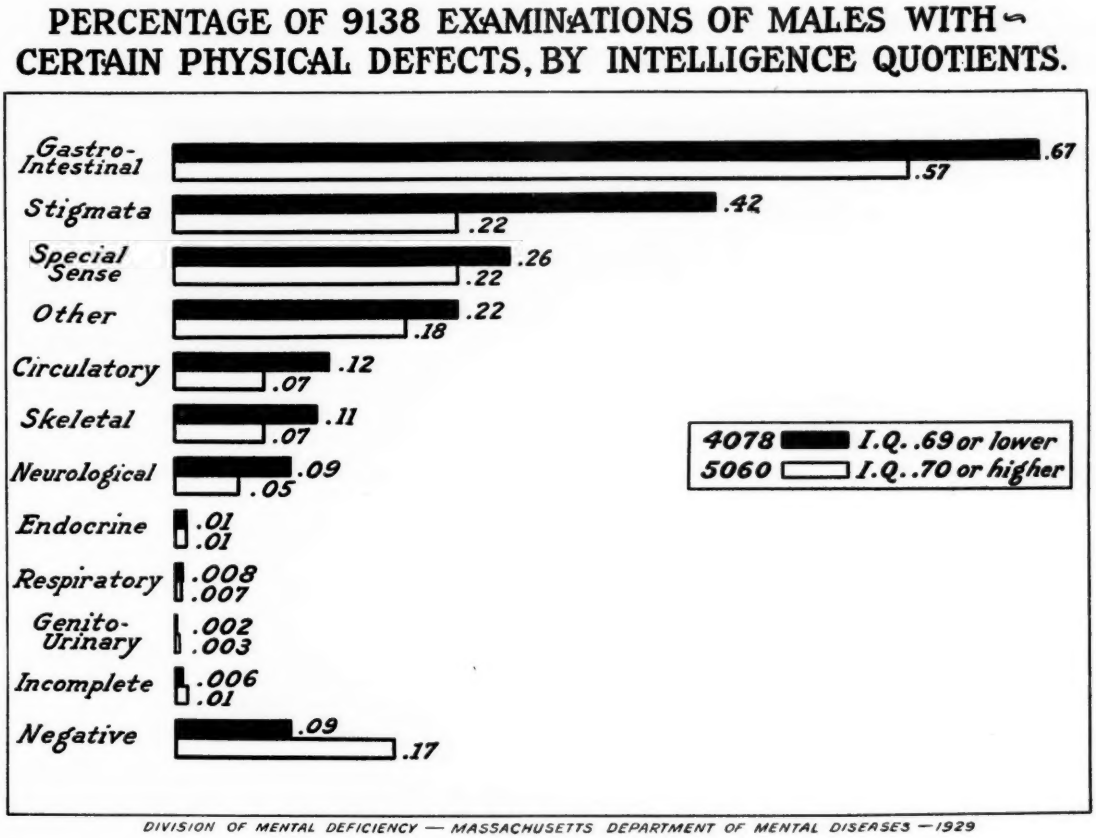
As in dealing with the weight factor, the cases showing certain physical defects were also divided into two groups:

I. Intelligence quotient .69 or lower.
II. Intelligence quotient .70 or higher.

The differences demonstrated in the males are shown in Graph VI and Table E, and for the females in Graph VII and Table F. It will be seen quite definitely that all physical defects exist to a larger extent in the group designated as feebleminded. For example, we observe that stigmata are twice as prevalent in the mentally defective as in those not mentally defective. When we come to the negative physical examinations, however, we see that there are, roughly, twice the number of negative physical examinations in the group with intelligence quotients *above* .70.

In considering the number of defects per child, we find that a sex difference is evident.

GRAPH VI


PERCENTAGE OF 9138 EXAMINATIONS OF MALES WITH CERTAIN PHYSICAL DEFECTS, BY INTELLIGENCE QUOTIENTS.
Gastro-Intestinal .67 .57
Stigmata .42 .22
Special Sense .26 .22
Other .22 .18
Circulatory .12 .07
Skeletal .11 .07
Neurological .09 .05
Endocrine .01 .01
Respiratory .008 .007
Genito-Urinary .002 .003
Incomplete .006 .01
Negative .09 .17
4078 I.Q. .69 or lower
5060 I.Q. .70 or higher
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4. The buccal cavity was added to the usual delineation of the gastro-intestinal system and consequently factors listed under this heading include dental defects.

TABLE E

FINDINGS OF PHYSICAL EXAMINATIONS OF 9138 MALES, RECORDED BY SYSTEMS IN ORDER OF FREQUENCY, BY INTELLIGENCE QUOTIENTS

| Defect | Total | I. Q. .69 or lower | I. Q. .70 or higher |
|---|---|---|---|
| Gastro-Intestinal | 5638 | 2737 | 2901 |
| Stigmata | 2862 | 1724 | 1138 |
| Special Sense | 2242 | 1079 | 1163 |
| Others | 1875 | 933 | 942 |
| Circulatory | 907 | 514 | 393 |
| Skeletal | 819 | 463 | 356 |
| Neurological | 685 | 395 | 290 |
| Endocrine | 119 | 51 | 68 |
| Respiratory | 71 | 35 | 36 |

GRAPH VII

## PERCENTAGE OF 5037 EXAMINATIONS OF FEMALES WITH CERTAIN PHYSICAL DEFECTS BY INTELLIGENCE QUOTIENTS.

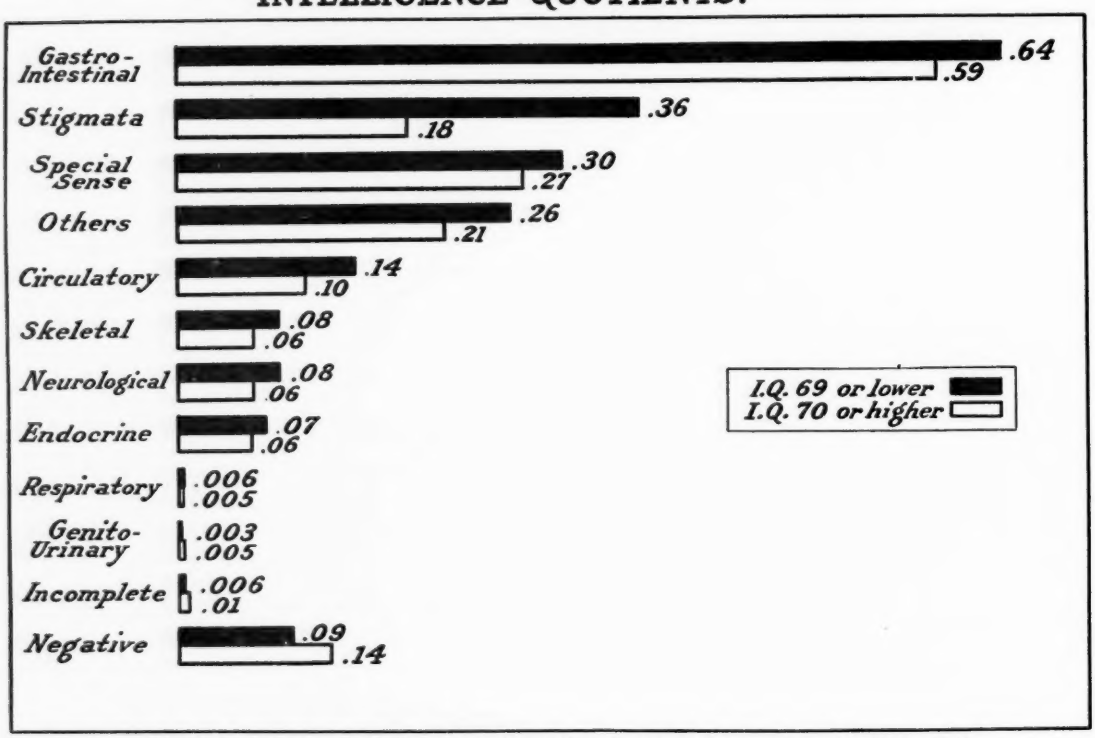


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TABLE F

FINDINGS OF PHYSICAL EXAMINATIONS OF 5037 FEMALES, RECORDED BY SYSTEMS IN ORDER OF FREQUENCY, BY INTELLIGENCE QUOTIENTS

| Defect | Total | I. Q. .69 or lower | I. Q. .70 or higher |
|---|---|---|---|
| Gastro-Intestinal | 3138 | 1776 | 1362 |
| Stigmata | 1477 | 1043 | 434 |
| Special Sense | 1458 | 832 | 626 |
| Others | 1220 | 724 | 496 |
| Circulatory | 638 | 391 | 247 |
| Skeletal | 385 | 236 | 149 |
| Neurological | 373 | 229 | 144 |
| Endocrine | 360 | 205 | 155 |
| Respiratory | 31 | 19 | 12 |
| Genito-Urinary | 22 | 9 | 13 |
| Incomplete | 46 | 18 | 28 |
| Negative | 601 | 260 | 341 |
| Total Defects | 9102 | 5464 | 3638 |
| No. of Examinations | 5037 | 2742 | 2295 |
| Defect per child* | 1.8 | 2.0 | 1.6 |

*Incomplete examinations excluded.

The girls have 1.8 defects per child while the boys have but 1.6 defects. At the same time, we notice material difference in defect per child when considered in relation to the I. Q. group. For the groups with intelligence quotients below .70, the girls show 2 defects per child, while the boys showed but 1.9. For the groups with intelligence quotients above .70, the girls showed 1.6 defects per child, while the score for the boys was 1.4. In general, we may say that if a child is below .70 in I. Q. he will average two physical defects, while the child with an I. Q. above .70 will average one and a half defects.

In summarizing the results, it appears that physical defect, in common with weight defect, is also associated with I. Q.'s below .70.

### AVERAGE INTELLIGENCE OF GROUPS *WITH* AND *WITHOUT* CERTAIN PHYSICAL DEFECTS

The rough association between physical defect and lower intelligence grouping which was revealed in the foregoing, warranted a more detailed investigation. For that reason we determined to calculate the mean intelligence of groups *with* certain defects, and then to compare these with the mean intelligence of groups *without* the same defects. This method would show whether or not a closer association was present. To demonstrate our method, we will follow through the procedure in dealing with skeletal defect.

From the physical examination we determined the number of children who could be considered as having a skeletal defect. We then determined the intelligence of the group *with* skeletal defect and compared this with the

GRAPH VIII

PERCENTAGE DISTRIBUTION OF
INTELLIGENCE QUOTIENTS OF GROUPS
WITH AND WITHOUT SKELETAL DEFECTS

TABLE G

DISTRIBUTION OF INTELLIGENCE QUOTIENTS WITH AND WITHOUT SKELETAL DEFECT, BY SEX

| I. Q. Groups | Total | 0-.49 | .50-.59 | .60-.69 | .70-.79 | .80-.89 | .90-.99 | 1.00+ | Mode | Median | Mean | S. D. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | *Male* | | | | | | | |
| With Defect | 819 | 68 | 133 | 262 | 222 | 95 | 20 | 19 | 60-69 | 67.9 | 67.9 | ± .47 |
| Without Defect | 8294 | 252 | 884 | 2454 | 2862 | 1235 | 440 | 167 | 70-79 | 71.9 | 72.0 | ± .13 |
| | | | | | *Female* | | | | | | | |
| With Defect | 385 | 35 | 69 | 132 | 103 | 35 | 8 | 3 | 60-69 | 66.7 | 66.4 | ± .64 |
| Without Defect | 4606 | 183 | 641 | 1664 | 1451 | 500 | 124 | 43 | 60-69 | 68.8 | 69.1 | ± .16 |

group *without* skeletal defect. The distributions are shown in Graph VIII and Table G. We see, for example, that the males without the defect have an average intelligence that is 4.1 points higher than those with skeletal defect. The females present a difference of 2.7 points in the intelligence scale. That is, in both sexes, the presence of a skeletal defect appeared to materially effect the mean intelligence quotients. If we look at the graphic distribution as outlined in Graph VIII, we see that the cases *without* defect are very much more in evidence in the 70-79, 80-89, 90+ groups, while the cases *with* skeletal defect are more in evidence in the lower groups. This graph clearly demonstrates the tremendous difference in our frequency distributions which was necessary to produce even such moderate disturbances in the mean I.Q. as 4.1 and 2.7 points.

The same method was applied to other defects. That we might not become involved in difficulties due to chance variations in small variations in small numbers, we selected only the first six physical defects, in order of their frequency. In Table H these distributions are indicated. In Graph IX we have simply recorded the mean intelligence quotients of the groups *with* and *without* specific defects. It will be observed that quite uniformly the cases *with* certain defects present a lower average intelligence than cases *without* the same defect. These differences vary from .9 points of the I.Q. scale, in special sense defect (female), to 7.4 points in stigmata (male). It is to be observed,

GRAPH IX

## DIFFERENCE BETWEEN MEAN INTELLIGENCE QUOTIENTS OF GROUPS WITH AND WITHOUT CERTAIN PHYSICAL DEFECTS

| DEFECT | SEX | WITH DEFECT | | | WITHOUT DEFECT | | | DIFFERENCE | $\sigma_d$ | DIFF. / $\sigma_d$ |
|---|---|---|---|---|---|---|---|---|---|---|
| | | N. | MEAN I.Q. | S.D. | N. | MEAN I.Q. | S.D. | | | |
| Stigmata | M | 2862 | 66.6 | ±.23 | 6251 | 74.0 | ±.14 | 7.4 | .26 | 28.6 |
| | F | 1477 | 63.9 | ± 29 | 3514 | 71.0 | ±.18 | 7.1 | .34 | 20.6 |
| Neurological | M. | 685 | 67.7 | ±.53 | 8428 | 72.0 | ±.13 | 4.3 | .54 | 7.9 |
| | F. | 373 | 66.6 | ±.64 | 4618 | 69.1 | ±.16 | 2.5 | .66 | 3.7 |
| Skeletal | M | 819 | 67.9 | ±.46 | 8294 | 72.0 | ±.13 | 4.1 | .47 | 8.7 |
| | F. | 385 | 66.4 | ±.64 | 4606 | 69.1 | ±.16 | 2.7 | .66 | 4.0 |
| Circulatory | M | 907 | 68.2 | ±.40 | 8206 | 72.0 | ±.13 | 3.8 | .42 | 9.0 |
| | F | 638 | 66.7 | ±.45 | 4353 | 69.2 | ±.17 | 2.5 | .48 | 5.2 |
| Gastro-Intestinal | M. | 5638 | 70.2 | ±.16 | 3475 | 73.9 | ±.21 | 3.7 | .26 | 14.2 |
| | F. | 3138 | 68.1 | ±.20 | 1853 | 70.3 | ±.27 | 2.2 | .33 | 6.6 |
| Special Sense | M. | 2242 | 70.5 | ±.26 | 6871 | 72.0 | ±.14 | 1.5 | .29 | 5.1 |
| | F. | 1458 | 68.3 | ±.30 | 3533 | 69.2 | ±.19 | .9 | .35 | 2.5 |

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TABLE H

DISTRIBUTION OF INTELLIGENCE QUOTIENTS IN GROUPS, BY SEX WITH AND WITHOUT CERTAIN PHYSICAL DEFECTS

MALE

| I. Q. Groups | Total | 0-.49 | .50-.59 | .60-.69 | .70-.79 | .80-.89 | .90-.99 | 1.00+ | Mode | Median | Mean | S. D. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *Stigmata* | | | | | | | | | | | | |
| With Defect | 2862 | 248 | 529 | 947 | 781 | 286 | 56 | 15 | 60-69 | 66.9 | 66.6 | ± .23 |
| Without Defect | 6251 | 72 | 488 | 1769 | 2303 | 1044 | 404 | 171 | 70-79 | 73.4 | 74.0 | ± .14 |
| *Neurological* | | | | | | | | | | | | |
| With Defect | 685 | 68 | 104 | 223 | 178 | 73 | 26 | 13 | 60-69 | 67.6 | 67.7 | ± .53 |
| Without Defect | 8428 | 252 | 913 | 2493 | 2906 | 1257 | 434 | 173 | 70-79 | 71.9 | 72.0 | ± .13 |
| *Skeletal* | | | | | | | | | | | | |
| With Defect | 819 | 68 | 133 | 262 | 222 | 95 | 20 | 19 | 60-69 | 67.9 | 67.9 | ± .47 |
| Without Defect | 8294 | 252 | 884 | 2454 | 2862 | 1235 | 440 | 167 | 70-79 | 71.9 | 72.0 | ± .13 |
| *Circulatory* | | | | | | | | | | | | |
| With Defect | 907 | 56 | 152 | 306 | 245 | 114 | 28 | 6 | 60-69 | 68.0 | 68.2 | ± .40 |
| Without Defect | 8206 | 264 | 865 | 2410 | 2839 | 1216 | 432 | 180 | 70-79 | 71.9 | 72.0 | ± .13 |
| *Gastro-Intestinal* | | | | | | | | | | | | |
| With Defect | 5638 | 240 | 690 | 1807 | 1895 | 751 | 205 | 50 | 70-79 | 70.4 | 70.2 | ± .16 |
| Without Defect | 3475 | 80 | 327 | 909 | 1189 | 579 | 255 | 136 | 70-79 | 73.5 | 73.9 | ± .21 |
| *Special Sense* | | | | | | | | | | | | |
| With Defect | 2242 | 103 | 291 | 685 | 723 | 300 | 98 | 42 | 70-79 | 70.5 | 70.5 | ± .26 |
| Without Defect | 6871 | 217 | 726 | 2031 | 2361 | 1030 | 362 | 144 | 70-79 | 71.9 | 72.0 | ± .14 |

FEMALE

| | Total | 0-.49 | .50-.59 | .60-.69 | .70-.79 | .80-.89 | .90-.99 | 1.00+ | Mode | Median | Mean | S. D. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *Stigmata* | | | | | | | | | | | | |
| With Defect | 1477 | 149 | 335 | 559 | 340 | 82 | 9 | 3 | 60-69 | 64.5 | 63.9 | ± .29 |
| Without Defect | 3514 | 69 | 375 | 1237 | 1214 | 453 | 123 | 43 | 70-79 | 70.6 | 71.0 | ± .18 |
| *Neurological* | | | | | | | | | | | | |
| With Defect | 373 | 30 | 67 | 132 | 102 | 30 | 8 | 4 | 60-69 | 66.8 | 66.6 | ± .64 |
| Without Defect | 4618 | 188 | 643 | 1664 | 1452 | 505 | 124 | 42 | 60-69 | 68.8 | 69.1 | ± .16 |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | *Skeletal* | | | | | | | |
| With Defect | 385 | 35 | 69 | 132 | 103 | 35 | 8 | 3 | 60-69 | 66.7 | 66.4 | ± .64 |
| Without Defect | 4606 | 183 | 641 | 1664 | 1451 | 500 | 124 | 43 | 60-69 | 68.8 | 69.1 | ± .16 |
| | | | | | *Circulatory* | | | | | | | |
| With Defect | 638 | 45 | 103 | 243 | 188 | 46 | 12 | 1 | 60-69 | 67.0 | 66.7 | ± .45 |
| Without Defect | 4353 | 173 | 607 | 1553 | 1366 | 489 | 120 | 45 | 60-69 | 68.9 | 69.2 | ± .17 |
| | | | | | *Gastro-Intestinal* | | | | | | | |
| With Defect | 3138 | 159 | 462 | 1155 | 995 | 284 | 67 | 16 | 60-69 | 68.2 | 68.1 | ± .20 |
| Without Defect | 1853 | 59 | 248 | 641 | 559 | 251 | 65 | 30 | 60-69 | 69.6 | 70.3 | ± .27 |
| | | | | | *Special Sense* | | | | | | | |
| With Defect | 1458 | 65 | 229 | 538 | 435 | 144 | 29 | 18 | 60-69 | 68.0 | 68.3 | ± .30 |
| Without Defect | 3533 | 153 | 481 | 1258 | 1119 | 391 | 103 | 28 | 60-69 | 69.0 | 69.2 | ± .19 |

however, that there is an absolute consistency in the result. That is, in the case of each defect, for both sexes, those without the defect are uniformly higher in I. Q. rating than those with the same defect. This uniformity in the twelve samples concerned is, in itself, of significance. The standard deviations of the differences between the mean intelligence quotients of groups *with* and *without* a specific defect have been computed. These differences for each defect were then tested for significance by the usual method of dividing the difference by the standard deviation of the difference. It will be noticed that the results vary from 2.5 in special sense defect (female) to 28.6 in the case of stigmata (male). If we accept three sigma as a criterion of significance, we can say that all but one of our results greatly exceed this figure.

In summarizing our inquiries into the relationship between the presence of physical defect and intelligence, we can state quite definitely that there is a positive association between the presence of any one of the physical defects enumerated and lower intelligence.

### AVERAGE INTELLIGENCE OF GROUPS WITH MULTIPLE PHYSICAL FACTORS

In the foregoing we have considered only the disturbance in mean intelligence in the presence of a single physical anomaly. It is evident that if we considered several physical defects together, the mean intelligence would be depressed to a far greater degree. It is hardly within the scope of this study to attempt to evaluate the various combinations of defects. This is an extremely complicated investigation and one

GRAPH X

**DIFFERENCE BETWEEN MEAN INTELLIGENCE QUOTIENTS OF GROUPS~**
**(1) WITH CERTAIN PHYSICAL DEFECTS AND UNDERWEIGHT.**
**(2) WITHOUT THE SAME PHYSICAL DEFECTS AND OVERWEIGHT.**

| DEFECT | SEX | UNDERWEIGHT WITH DEFECT | | | OVERWEIGHT WITHOUT DEFECT | | | DIFFERENCE | $\sigma_d$ | DIFF. / $\sigma_d$ |
|---|---|---|---|---|---|---|---|---|---|---|
| | | N. | MEAN I.Q. | S.D. | N. | MEAN I.Q. | S.D. | | | |
| Stigmata | M. | 1118 | 63.7 | ±.35 | 1488 | 75.4 | ±.31 | 11.7 | .46 | 25.4 |
| | F. | 593 | 62.4 | ±.45 | 958 | 72.4 | ±.35 | 10.0 | .56 | 17.8 |
| Neurological | M. | 242 | 63.8 | ±.83 | 1933 | 73.5 | ±.27 | 9.7 | .82 | 11.8 |
| | F. | 137 | 65.5 | ±.99 | 1210 | 70.6 | ±.33 | 5.1 | 1.00 | 5.1 |
| Skeletal | M. | 345 | 65.1 | ±.69 | 1970 | 73.5 | ±.27 | 8.4 | .74 | 11.3 |
| | F. | 165 | 64.6 | ±.95 | 1239 | 70.4 | ±.33 | 5.8 | 1.00 | 5.8 |
| Circulatory | M. | 337 | 65.7 | ±.62 | 1858 | 73.7 | ±.28 | 8.0 | .68 | 11.7 |
| | F. | 248 | 64.1 | ±.72 | 1158 | 70.7 | ±.35 | 6.6 | .80 | 8.2 |
| Gastro-Intestinal | M. | 2058 | 67.1 | ±.25 | 879 | 74.8 | ±.43 | 7.7 | .49 | 15.7 |
| | F. | 1099 | 65.7 | ±.32 | 536 | 71.7 | ±.52 | 6.0 | .62 | 9.6 |
| Special Sense | M. | 737 | 66.7 | ±.42 | 1614 | 73.4 | ±.30 | 6.7 | .51 | 13.1 |
| | F. | 448 | 65.7 | ±.52 | 922 | 70.5 | ±.40 | 4.8 | .66 | 7.2 |

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TABLE J

DISTRIBUTION OF INTELLIGENCE QUOTIENTS OF GROUPS, BY SEX:
(1) WITH CERTAIN PHYSICAL DEFECTS AND UNDER-WEIGHT,
(2) WITHOUT THE SAME DEFECT AND OVER-WEIGHT.

MALE

| I. Q. Groups | Total | 0-.49 | .50-.59 | .60-.69 | .70-.79 | .80-.89 | .90-.99 | 1.00+ | Mode | Median | Mean | S. D. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *Stigmata* | | | | | | | | | | | | |
| With Defect and Under Weight | 1118 | 131 | 246 | 411 | 252 | 65 | 11 | 2 | 60-69 | 64.4 | 63.7 | ± .35 |
| Without Defect and Over Weight | 1488 | 13 | 98 | 388 | 519 | 292 | 117 | 61 | 70-79 | 74.7 | 75.4 | ± .31 |
| *Neurological* | | | | | | | | | | | | |
| With Defect and Under Weight | 242 | 35 | 44 | 89 | 56 | 12 | 6 | 0 | 60-69 | 64.7 | 63.8 | ± .83 |
| Without Defect and Over Weight | 1933 | 32 | 183 | 541 | 665 | 333 | 120 | 59 | 70-79 | 73.1 | 73.5 | ± .27 |
| *Skeletal* | | | | | | | | | | | | |
| With Defect and Under Weight | 345 | 38 | 69 | 122 | 80 | 27 | 6 | 3 | 60-69 | 65.4 | 65.1 | ± .69 |
| Without Defect and Over Weight | 1970 | 38 | 181 | 558 | 676 | 334 | 124 | 59 | 70-79 | 73.0 | 73.5 | ± .27 |
| *Circulatory* | | | | | | | | | | | | |
| With Defect and Under Weight | 337 | 22 | 71 | 135 | 79 | 22 | 6 | 2 | 60-69 | 65.6 | 65.7 | ± .62 |
| Without Defect and Over Weight | 1858 | 32 | 160 | 527 | 647 | 309 | 119 | 64 | 70-79 | 73.2 | 73.7 | ± .28 |
| *Gastro-Intestinal* | | | | | | | | | | | | |
| With Defect and Under Weight | 2058 | 130 | 336 | 769 | 601 | 180 | 36 | 6 | 60-69 | 67.3 | 67.1 | ± .25 |
| Without Defect and Over Weight | 879 | 11 | 79 | 243 | 271 | 161 | 69 | 45 | 70-79 | 73.9 | 74.8 | ± .43 |
| *Special Sense* | | | | | | | | | | | | |
| With Defect and Under Weight | 737 | 50 | 124 | 284 | 205 | 61 | 12 | 1 | 60-69 | 66.8 | 66.7 | ± .42 |
| Without Defect and Over Weight | 1614 | 34 | 143 | 473 | 536 | 274 | 104 | 50 | 70-79 | 72.9 | 73.4 | ± .30 |

FEMALE

| I. Q. Groups | Total | 0-.49 | .50-.59 | .60-.69 | .70-.79 | .80-.89 | .90-.99 | 1.00+ | Mode | Median | Mean | S. D. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *Stigmata* | | | | | | | | | | | | |
| With Defect and Under Weight | 593 | 69 | 146 | 242 | 110 | 25 | 1 | 0 | 60-69 | 63.3 | 62.4 | ± .45 |
| Without Defect and Over Weight | 958 | 16 | 79 | 310 | 357 | 135 | 41 | 20 | 70-79 | 72.0 | 72.4 | ± .35 |
| *Neurological* | | | | | | | | | | | | |
| With Defect and Under Weight | 137 | 13 | 23 | 50 | 43 | 6 | 2 | 0 | 60-69 | 66.6 | 65.5 | ± .99 |
| Without Defect and Over Weight | 1210 | 42 | 134 | 406 | 421 | 145 | 42 | 20 | 70-79 | 70.5 | 70.6 | ± .33 |
| *Skeletal* | | | | | | | | | | | | |
| With Defect and Under Weight | 165 | 18 | 33 | 60 | 40 | 12 | 1 | 1 | 60-69 | 65.3 | 64.6 | ± .95 |
| Without Defect and Over Weight | 1239 | 42 | 145 | 417 | 424 | 149 | 41 | 21 | 70-79 | 70.3 | 70.4 | ± .33 |
| *Circulatory* | | | | | | | | | | | | |
| With Defect and Under Weight | 248 | 26 | 52 | 90 | 67 | 12 | 1 | 0 | 60-69 | 65.1 | 64.1 | ± .72 |
| Without Defect and Over Weight | 1158 | 42 | 130 | 376 | 401 | 145 | 43 | 21 | 70-79 | 70.7 | 70.7 | ± .35 |
| *Gastro-Intestinal* | | | | | | | | | | | | |
| With Defect and Under Weight | 1099 | 71 | 206 | 459 | 293 | 59 | 11 | 0 | 60-69 | 65.9 | 65.7 | ± .32 |
| Without Defect and Over Weight | 536 | 16 | 56 | 178 | 165 | 83 | 23 | 15 | 70-79 | 71.0 | 71.7 | ± .52 |
| *Special Sense* | | | | | | | | | | | | |
| With Defect and Under Weight | 448 | 27 | 100 | 167 | 119 | 29 | 5 | 1 | 60-69 | 65.8 | 65.7 | ± .52 |
| Without Defect and Over Weight | 922 | 34 | 109 | 305 | 310 | 114 | 36 | 14 | 70-79 | 70.4 | 70.5 | ± .40 |

which will be left for the future. However, it was determined to investigate the intelligence associated with the presence of a single defect and also the factor of under-weight. That is, we studied the intelligence of groups *with specific physical defects who were also under-weight* and compared their intelligence with that of groups *without the same physical defect who were over-weight*. This gave us the two extremes. This method was applied quite uniformly in the case of each of the six physical defects previously studied. The results are shown in Graph X and Table J.

It will be remembered that when we were considering the presence or absence of the factor of under-weight we found a difference of five points in the I. Q. scale for males and four points for females. When we considered the various physical defects we found the differences in I. Q. ranging from 1.9 (special sense, females) to 7.4 (stigmata, males). However, when we make a combination and consider the two factors of physical defect and under-weight we notice (Graph X) that the differences now vary between 4.8 in special sense plus under-weight (females) to 11.7 in stigmata plus under-weight (males).

Thus when we consider the combination of two physical anomalies we get a result which shows an even greater association between physical defects and the lower grades of intelligence. We have not attempted to discuss whether or not the factor of under-weight necessarily accompanies certain physical defects. In brief we can say that the factors tend to be associated, but our selves and not simply upon the mere presence or absence of a defect.

To carry this still further we continued our investigation and studied the following groups:

I. Cases with negative physical examinations and over-average in weight.

II. Cases with one or more physical defects and under-average in weight.

TABLE K

DISTRIBUTION OF INTELLIGENCE QUOTIENTS OF GROUPS:
(1) WITH ONE OR MORE PHYSICAL DEFECTS,
(2) WITH NEGATIVE PHYSICAL EXAMINATION.

| I. Q. Groups | Total | .0-.49 | .50-.59 | .60-.69 | .70-.79 | .80-.89 | .90-.99 | 1.00-1.09 | 1.10-1.19 | 1.20+ | Mode | Median | Mean | S. D. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| (1) With Defect or Defects | M. 7840 | 314 | 958 | 2410 | 2602 | 1074 | 345 | 86 | 26 | 25 | 70-79 | 70.9 | 70.9 | ± .14 |
| | F. 4436 | 212 | 654 | 1616 | 1369 | 435 | 109 | 33 | 6 | 2 | 60-69 | 68.3 | 68.5 | ± .17 |
| (2) Negative Physical Examination | M. 1298 | 8 | 65 | 323 | 482 | 256 | 115 | 36 | 7 | 6 | 70-79 | 75.2 | 76.1 | ± .32 |
| | F. 601 | 11 | 58 | 191 | 199 | 103 | 30 | 5 | 4 | 0 | 70-79 | 72.0 | 72.4 | ± .47 |

TABLE L

DISTRIBUTION OF INTELLIGENCE QUOTIENTS OF GROUPS:
(1) OVER-AVERAGE WEIGHT AND WITH NEGATIVE PHYSICAL EXAMINATION,
(2) UNDER-AVERAGE WEIGHT AND WITH ONE OR MORE PHYSICAL DEFECTS.

| I. Q. Groups | Total | 0-.49 | .50-.59 | .60-.69 | .70-.79 | .80-.89 | .90+ | Mode | Median | Mean | S. D. |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. *Over-Weight and Negative Examination* | | | | | | | | | | | |
| Male | 338 | 2 | 13 | 94 | 106 | 69 | 54 | 70-79 | 75.6 | 76.9 | ± .66 |
| Female | 178 | 2 | 14 | 51 | 60 | 37 | 14 | 70-79 | 73.6 | 74.0 | ± .87 |
| 2. *Under-Weight and One or More Physical Defects* | | | | | | | | | | | |
| Male | 2714 | 159 | 436 | 994 | 824 | 231 | 70 | 60-69 | 67.6 | 67.5 | ± .22 |
| Female | 1493 | 95 | 289 | 603 | 398 | 90 | 18 | 60-69 | 66.0 | 65.8 | ± .27 |

figures do not justify a dogmatic conclusion that this association is of greater significance in one defect than in another.

### AVERAGE INTELLIGENCE OF GROUPS WITH NEGATIVE PHYSICAL EXAMINATION

It was thought that possibly the negative physical examinations might have contained a preponderance of higher grade cases, and thus have influenced our results unduly. We therefore investigated the intelligence distribution of cases with *one or more physical defects* and compared it with the intelligence of cases with a *negative physical* examination. The results are seen in Table K. We see here differences of 5.2 points in the I. Q. scale for males, and 3.9 points for females. This does not give the appearance of an abnormal grouping of high grade cases in the negative physical examination. It rather points out the fact that our groups with and without certain defects are not wholly dependent upon the negative group for the differences in the mean I. Q.'s which are evident. In other words, some of the differences must depend upon the quality of the defects themThe distribution of intelligence quotients of these groups is shown in Table L. The mean differences in the I. Q. scale are 8.0 for the males and 7.6 points for the females. In this table we see no new findings but simply an elaboration of facts previously enumerated. Again we may say that the presence of physical defects and the factor of under-weight are definitely associated with the lower grades of intelligence.

### DISCUSSION

In discussing the significance of our data, we find ourselves in a difficult situation. There are so many possibilities in the interpretation of these results that a complete consideration would involve points which are beyond the scope of this paper. Consequently there will be a brief discussion of the extreme viewpoints which might be advanced, with a reservation on the part of the writer that the final answer probably lies midway between these two extremes. The two opposed viewpoints may be recorded as follows:

I. That the physical defects observed in retardates influence the degree of the mental defect.

II. That mental defect is an indication of biological defectiveness on the part of the individual, and that the more severe grades of this defect will show evidence of defectiveness in other spheres.

In considering the first point we would have to assume that the observed deviation from normal intelligence was caused by the physical defect. In other words, we would assume that these mental defectives had limited intelligences to begin with and, owing to the presence of physical defect, the intelligence was depressed an additional few points of the I.Q. scale. To use a single case, for example, we might say that a certain child would have had an intelligence quotient of .68, but owing to the handicap of physical defects, it was 8 points lower, or .60.

If we consider the soma as the home of the intelligence, we see that there is some reason for the thought that the intelligence may be influenced by an abnormal or markedly defective physical environment. This theory would hold that a defective intelligence was so influenced by the defective body that it could not attain the level justified by its germ plasm. We can understand that a serious physical defect, such as a poor heart, a kyphosis or defective vision or hearing, would materially interfere with the reception and retention of experience on the part of the child. In this manner it is not inconceivable that such an interference should be reflected in the intelligence quotient, particularly as the latter is essentially based on recalled experience.

However, this theory receives a decided setback when we study the order of importance of the physical defects as seen in Graph IX. There we observe that the presence of serious defects such as those of vision and hearing (special sense) is associated with the *least* difference in the intelligence. On the other hand, the presence of stigmata, which certainly cannot be considered as a material physical hindrance, is associated with the *greatest* difference in the intelligence. Here we have the paradoxical situation that the more serious the physical defect, the less the effect on the intelligence. The less serious the physical defect the more marked is the associated mental difference. It appears, then, that it is not a quantitative but a qualitative measure that we need. Are there some hidden qualities in stigmata, defective tonsils and teeth, or neurological defects which make them more undesirable to already enfeebled mental processes?

In considering the other extreme of this theoretical discussion of the problem, we reverse our reasoning. We no longer consider physical defects as causes of the mental deviations with which they are associated, but as results. Certainly we have received this suggestion from the fact that the greatest depression of the intelligence was associated with the so-called "stigmata of degeneration". This fact suggests that mental defect is a condition that is associated with a definite defectiveness of the germ plasm. For example, if we consider mental deficiency as a stigma in the field of intelligence, so we may consider neurological defect as a stigma in the anatomical field. In this light it becomes increasingly difficult to consider the possibility of one defect as the cause of another. It seems more logical to consider them in the light of *association.* If we have one defect in mild form we may have another; however, if our initial defect is in severe form, we may expect to see defectiveness in other fields in the form of other quite unrelated defects. If we exclude the pathological types, we may say that mental defect is simply an indication of a biological defectiveness of the individual confined to the field of intelligence. Then the lower grades of mental defect indicate a greater degree of this biological defectiveness. When we find this greater degree of defectiveness we must expect to see not a single defect but multiple defects in many other fields.

The uniformity of our results for all defects makes us believe that all physical defects have approximately equal ratings as indicators of more advanced mental defectiveness. That is, the presence of any one of them in a group is associated with a definite depression of the intelligence of that group. The lowering of the mean intelligence when associated with stigmata means simply that the "stigmata of degeneration" are indicators of a more pronounced germinal defect which has been reflected in the lower intelligence. If the fact that an individual has a low intelligence and also stigmata means that he possesses a more defective germ plasm, may we not apply the same conclusion to situations in which the individual has low intelligence and *other* physical defects? Is every physical defect an anatomical stigma? Are all physical defects indicators of germinal degeneration in the anatomical field?

The present study rather bears out the viewpoint of theory II. However, it is not thought that this theoretical discussion begins to meet the problem in any comprehensive way. We have merely mentioned a few of a large number of related facts. These are so closely interwoven that it is almost impossible to separate them into their component parts. We can say, however, that study of this material reveals a *tendency* for individuals, defective in the sphere of intelligence, to accumulate defects in other spheres in proportion to the severity of the mental defect. In the field of eugenics this is of signal importance. The difficulty of disposing of a germ plasm which is defective in many characteristics is self-evident. Do processes of a

degenerative nature accumulate in germ plasm until the last individual is overwhelmed, dies, and thus removes the danger to the race? Again we can see that this removal would be a very quick process if man were still battling with the other animals for survival. With present day civilization, where it is almost impossible for infant, child or adult to die, no matter how defective, the removal does not take place. Is civilization the incubator of defective germ plasm?

SUMMARY

I. The findings in the physical examinations of 14,379 retarded public school children are reviewed.

II. The mean intelligence quotient of the males was found to be .71, that of the females .69.

III. Under-average weight is more characteristic of children with an intelligence quotient of .69 or lower. It is not as characteristic of children with an I. Q. of .70 or higher.

IV. There is no difference between the mean intelligence quotients of groups over-average or average in weight. The mean intelligence of groups *average in weight* exceeds that of groups *under-average in weight* by 5.4 points of the I. Q. scale for males, and 4.3 points for females.

V. Physical defects appear to be more characteristic of the groups with intelligence quotients .69 or lower than they are of groups with intelligence quotients .70 or higher. The negative physical examinations are twice as frequent in the group with intelligence quotients .70 or higher.

VI. The mean intelligence of groups *with* any one of the six defects studied was found to be *lower* than the mean intelligence of groups *without* the same defect. These differences in points of the I. Q. scale, varied from .9 in special sense (females) to 7.4 in stigmata (males). The differences are greater for males than females in each defect.

VII. The mean intelligence quotients of groups *with certain physical defects and under-weight* were found to be *lower* than that of other groups *without the same defect and over-weight.* Differences in points of the I. Q. scale in this case varied from 4.8 in special sense (females) to 11.7 in stigmata (males). Again the differences for each combination of defects were greater for the males.

VIII. The mean intelligence of groups *with one or more physical defects* was found to be *lower* than that of groups *with negative physical examination* by 5.2 points of the I. Q. scale for males and 3.9 points for females.

IX. The mean intelligence of groups *with one or more physical defects and under-weight* was found to be *lower* than that of groups *with negative physical examinations and over-weight.* Differences in points of the I. Q. scale were 8.0 for males and 7.6 for females.

X. This investigation demonstrates a positive association between the factors of physical defect or under-weight and the *lower* levels of intelligence. The lowering of the mean intelligence associated with these factors is more marked in the case of males than females. This is true for each defect and each combination of defects.

## THE CARE OF THE SURGICAL DIABETIC

BY W. RICHARD OHLER, M.D.*

THE thoughts expressed in this brief communication are the result of a study of the records of cases of surgical diabetes at the Boston City Hospital for a five year period before and a five year period after the introduction of insulin. The purpose of this paper is not to present statistics or to analyze cases or results of various kinds of surgical treatment, but simply to present a few ideas and to suggest some simple rules of procedure. Before presenting any definite ideas on the subject, it should be stated that this study is made from the records of a large general hospital, divided into separate distinct services, with a resulting lack of unanimity in the treatment of any given surgical condition. The thoughts which appear worthy of comment may be briefly enumerated as follows:—

1. The surgical diabetic should always be looked upon as a surgical emergency because generally a surgical diabetic is a severe diabetic and in diabetes, sepsis travels rapidly and surgical symptoms are often obscured.

2. The presence of sugar or diacetic acid in the urine is no contra-indication to surgery. Sound surgical judgment need never be set aside because of diabetes, regardless of the severity of the diabetes, provided prompt and adequate medical treatment is given before and after operation. This is especially true in regard to conditions requiring drainage of septic areas.

3. The presence of pus always increases the severity of the diabetes. A study of records proves unmistakably that the septic case allowed to wait for surgical intervention until the patient's general condition improves, always dies.

4. Even the case of general septicemia may be kept sugar free with the aid of insulin, but insulin is of no help in halting the spread of the infection.

*For record and address of author see "This Week's Issue," page 282.

5. Every septic wound in a diabetic should be seen every day by the visiting surgeon. The carefully bandaged mildly gangrenous toe may become a septic foot and leg within twenty-four or forty-eight hours.

6. Beginning coma in a non surgical diabetic may present the symptoms of an acute surgical abdomen. In diabetic coma the skin is always dry and the patient nearly always has nausea and vomiting and a leucocytosis.

7. That case does best where there is complete co-öperation between surgeon and internist. This co-öperation falls short of the ideal unless the internist sees the patient before operation and unless daily visits—preferably combined can be made at least during the critical period of illness.

8. How such co-öperation can be accomplished in the large general hospital where both the medical and surgical sides are divided into several separate services, each with a separate corps of visiting physicians and internes, constitutes a problem which deserves much more attention than is apparent at present.

9. The most important single service in the proper care of the surgical diabetic is to supply the patient with a competent special nurse, or to detail one of the ward nurses to the special care of the patient.

10. Provision should be made for frequent testing of the urine for sugar and diacetic acid on the ward—at least during the critical period of illness. This is of much greater value than collecting a twenty-four hour amount of urine and waiting until the next day for the report.

11. Blood sugar determinations are of general value and should be made as often as indicated. However, a blood sugar determination is of no particular value in helping to determine the kind and the time of surgical procedure. Frequently valuable time has been lost in waiting for a blood sugar report.

12. The proper dose of insulin can be safely regulated by testing the urine shortly before each meal and by giving insulin according to some sort of color scheme. A suggested plan is as follows:—

| | |
|---|---|
| No sugar, | no insulin |
| Greenish color, | 5 units |
| Yellow green color, | 10 units |
| Brown color, | 15 units |
| Red color, | 20 units |
| Diacetic acid, | 20 units |

Mention has been made of the difficulty of establishing any set rule or procedure for the treatment of any given surgical condition in a large general hospital. Experience has taught us, however, that in the treatment of a condition like surgical diabetes, a definite plan of campaign with its resulting co-öperation and team work, does give best results. The following rules are presented, not with the idea that they will cover every case, or that the rules may not be changed from time to time to advantage, but with the hope that these definite suggestions may serve as a guide to the surgeon and to the residents and internes who have direct charge of the patient.

Pre-operative Rules:—

Where the operation is one of choice.

1. Patient under complete care of internist.

2. Patient sugar free and blood sugar below 250 mgm. on a diet of about 100 gms. of C-h, adequate protein and fat. (Protein 1 gm. per kg.)

3. On day of operation patient to receive from 30 to 50 grams of glucose in the form of oatmeal gruel or orange juice, at least two hours before operation.

4. If patient is receiving insulin, morning dose of insulin to be given as usual—at time of glucose meal.

Where the operation is one of necessity.

1. Test urine for sugar and diacetic acid and give insulin accordingly,—the amount to be regulated according to the scheme outlined above.

2. Before operation introduce carbohydrate by some means. A carbohydrate meal—glucose by rectum or intravenously—30 to 50 grams of glucose desirable.

Operative Rules:—

The same for operations of choice and for operations of necessity.

1. During operation prevent heat and fluid loss as much as possible. Keep patient warm. Keep patient covered.

2. In the severe case, it is desirable to give 1000 c.c. of saline solution subcutaneously before patient leaves the operating table.

Post-operative Rules:—

The same for operations of choice or necessity.

1. Immediately after operation, start rectal tap water.

2. Test urine within first hour and give insulin if necessary. (In some clinics 5 or 10 units of insulin are given empirically before the patient leaves the operating room.)

3. Test urine every two hours subsequently for first 24 hours—giving insulin whenever necessary. Test urine every four hours during second day. Test urine every six hours during third day—which means three times a day before meals and at 10 P. M. Do not become alarmed over the appearance of sugar, but watch carefully for the appearance of diacetic acid.

4. Start fluids by mouth as soon as possible. Oatmeal gruel, ginger ale, milk, may be given ad. lib. for first 24 hours. If possible give patient 1000 c.c. of fluids during the first 24 hours.

5. If fluids cannot be taken by mouth, give

carbohydrate in the form of glucose by rectum or intravenously. Patient to get at least 50 grams of glucose in first 24 hours.

6. Often, especially in abdominal and throat cases, it is advisable to allow the surgeon to plan the diet entirely and to control the diabetes by insulin.

SUMMARY

As a result of a study of the records of cases of surgical diabetes in a large general hospital, certain ideas are presented and some simple rules of procedure suggested with the hope that they may be helpful in the proper management of the above condition.

# NUTRITIONAL ANEMIA IN INFANCY:

## A Deficiency Disease*

BY LEWIS WEBB HILL, M.D.†

### DEFINITION

NUTRITIONAL anemia in infants is a secondary disease, occurring usually between the ages of six months to two years, dependent, probably, upon errors of nutrition.

### SYMPTOMS

The chief sign is, of course, pallor, which is usually marked. Occasionally there may be a yellowish tinge to the skin, such as is seen in some of the hemolytic anemias, but this is uncommon. The spleen and liver are not usually enlarged. The weight of the child may be normal in many cases, and it is plump and fat, but the tissues are always soft and flabby. Rickets may or may not be associated with this type of anemia. In our series of 28 patients there were few who showed gross signs of it.

The blood picture is characteristic, and varies little. The hemoglobin is reduced, usually more than the red cells: that is, it is a chlorotic type of anemia. The lowest hemoglobin percentage that we have seen is 21% (in this case the red blood count was 3,700,000), the lowest red count 2,000,000. The red blood cells show a good deal of achromia, but only very moderate variations in size and shape. We have rarely noted nucleated red cells, stippling or polychromatophilia. The white blood cells show nothing noteworthy, either qualitatively or quantitatively.

### OCCURRENCE

Nutritional anemia occurs especially in premature infants, twins, and in infants who have been kept too long on an exclusive milk diet. It is not common before the age of 9 months or after 2 years. The youngest case in our series was aged 2 months, the oldest 3 years and 4 months. Of our 28 cases, 8 were twins, 8 prematures, 9 were full term infants who had been kept too long on an exclusive milk diet, and in 3 cases the etiology was undetermined.

The following table shows the age, hemoglobin percentages, red count, and apparent etiology, of the cases in our series:

TABLE I

| No. | Age | Hgb. | R. B. C. | Apparent Etiology |
|---|---|---|---|---|
| 1. | 5 mos. | 64 | 3,120,000 | Twin |
| 2. | 5 mos. | 63 | 3,600,000 | Twin |
| 3. | 23 mos. | 43 | 2,952,000 | Milk Diet |
| 4. | 2 mos. | 54 | 3,216,000 | |
| 5. | 4½ mos. | 52 | 3,500,000 | Twin |
| 6. | 19 mos. | 41 | 4,600,000 | Milk Diet |
| 7. | 7 mos. | 21 | 3,700,000 | Twin |
| 8. | 13 mos. | 31 | 5,616,000 | Premature |
| 9. | 8 mos. | 27 | 3,900,000 | Premature |
| 10. | 11 weeks | 67 | 4,300,000 | |
| 11. | 13 mos. | 38 | | |
| 12. | 14 mos. | 35 | 5,600,000 | Premature |
| 13. | 14 mos. | 57 | | Premature twin; milk diet |
| 14. | 14 mos. | 40 | | Premature twin; milk diet |
| 15. | 20 mos. | 32 | 2,656,000 | Premature; milk diet |
| 16. | 21 mos. | 50 | 2,000,000 | Milk diet |
| 17. | 17 mos. | 60 | 4,200,000 | Twin |
| 18. | 17 mos. | 70 | 4,000,000 | Twin |
| 19. | 11 mos. | 55 | 2,700,000 | |
| 20. | 19 mos. | 44 | 4,656,000 | Milk diet |
| 21. | 17 mos. | 36 | 3,724,000 | Premature |
| 22. | 17 mos. | 46 | 4,110,000 | |
| 23. | 11 mos. | 35 | 4,320,000 | Milk diet |
| 24. | 27 mos. | 38 | 4,650,000 | Milk diet |
| 25. | 14 mos. | 35 | 2,352,000 | Milk diet |
| 26. | 2 7/12 yrs. | 31 | 3,416,000 | Milk diet |
| 27. | 3 4/12 yrs. | 25 | 3,660,000 | Milk diet |
| 28. | 16 mos. | 40 | 3,800,000 | Premature; milk diet |

### ETIOLOGY AND PATHOGENESIS

We consider this type of anemia to be a definite deficiency disease, comparable to rickets and scurvy, dependent upon a lack of iron and possibly pigment in the diet, and that it can be almost always clearly differentiated from other forms of anemia seen in infancy. The question whether it bears any relationship to the severe types of anemia with enlarged spleen (such as the so-called von Jaksch's anemia, or "splenic anemia") is an interesting and difficult one to answer. It seems to us that it does not, and that these anemias belong in a different category, and have a different etiology, at present unknown. Their clinical picture is not the same, the blood findings are entirely dissimilar, and they are not so likely to respond to the

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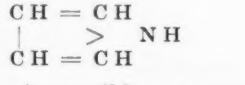
†For record and address of author see "This Week's Issue," page 282.

therapy to which simple nutritional anemia responds so well. It may be argued that splenic anemia, with many nucleated red cells, changes in the white cells and so on, is simply a severe nutritional anemia, modified by repeated infections of one sort or another, or by rickets. We can only say that we have seen many patients with severe nutritional anemias with the hemoglobin as low as 25%, who have had rickets, and have had repeated infections, and we have not seen the spleen or the blood take on the characteristics of "von Jaksch's anemia." The former is essentially a disturbance of hemoglobin synthesis, and responds well when the proper materials for forming hemoglobin are furnished; the latter is a much more profound and uncertain condition, with far reaching bone marrow changes, and responds very uncertainly to therapy of any sort.

There is much evidence to support the thesis that nutritional anemia is a relatively simple deficiency disease; so much, in fact, that it is difficult to present it in a clear manner.

In the first place, it may be said that we are dealing with an anemia the chief characteristic of which is deficient hemoglobin formation. Let us consider for a moment the substance, hemoglobin. It is a definite chemical compound, consisting of a pigment, hematin, iron, and a protein (globin), and cannot be synthesized unless a sufficient amount of each of its constituent parts is present. The pigment is supplied to the infant from the yellow pigment of milk fat, consisting of carotins and xanthophylls, substances derived originally from chlorophyll and other plant pigments. These pigments and hematin are closely related, in that they contain the pyrrol ring, an organic radicle with the formula



it is probably impossible to synthesize this nucleus in the body, and it must be supplied in the form of pigment-containing foods, such as the fat of milk, egg yolk, vegetables, or meat. There is some re-absorption of pigment liberated by the breaking down of blood cells, but a considerable amount is lost each day in the feces in the form of bile pigment, so that it might be easy, in the case of an infant over eight or nine months of age, who is taking an exclusive milk diet, for a negative pigment balance to result.

Hemoglobin contains only 0.3% of iron. The iron needs of the body are therefore small, but none the less important. The young infant derives its iron from the very small quantities of iron in milk (0.55 mgm per litre in human milk, 0.17 mgm. per litre in cow's milk) and from its own reserve supply in the liver; the older infant is supplied by the iron-containing foods, such as meat, vegetables, egg yolk, and prunes. Bunge[1] found, in his classical work in 1889, that the young of mammals are born with a reserve of iron in the liver, to last until they are able to take iron-containing food, and that the amount of this iron deposit varies directly with the duration of lactation. The liver of a newly born infant contains, proportionately at least, three times as much iron as the adult liver. Most of this is deposited in the last months of pregnancy, and Hougounenq[2] found that there was 0.421 gm. of $Fe_2O_3$ in the body of a full term foetus, and only 0.119 gm. in that of a six-months foetus.

A number of investigations have been carried out, in which it has been shown that it is possible to produce a high degree of anemia in young growing animals by an iron-poor diet.[3] It is not possible to do this in adult animals, however, nor do we see anemia in conditions of starvation, such as pyloric stenosis, or "athrepsia" from other causes: there is rather an atrophy of the whole blood. This means that growth is necessary to produce the type of anemia we are discussing, and the more rapid the growth, the more rapid the development of the anemia. In this, nutritional anemia is analogous to rickets.

In the infant, iron, as protein, must be furnished not only to supply the daily loss from the body in the feces, but also to allow for growth. The iron needs are difficult to calculate, and only an approximate idea may be arrived at. We calculated, some time ago,[4] that a year-old infant needs, roughly, about two milligrams of iron a day. This is not obtained with a milk diet.

It has been thought (Kleinschmidt, Czerny) that nutritional anemia could not be explained by a simple deficiency hypothesis, and that there was an active injury to the red blood cells by the fatty acids of milk. This theory is rendered improbable by the more recent work of Denzer, Herman, and Bass,[5] who showed that there is no increase of urobilin in the stools, and hence presumably no increased blood destruction.

## TREATMENT

It has been known for a long time that the addition of such foods as eggs, vegetables, and meat to the diet, would gradually bring the blood back to normal. In this, we are not particularly interested, but are especially concerned with the individual factors which are responsible. What food will *best* furnish the materials from which to synthesize hemoglobin? What is the value of inorganic iron? What mode of feeding or medicinal therapy, or a combination of the two, will bring about the most rapid results?

There has been, for many years, a discussion as to whether inorganic iron preparations can be utilized in the formation of hemoglobin; some observers have insisted that they can, while others have been equally convinced that

it is necessary to supply iron "from the market, and not from the pharmacy" in order to get results. It seems to us that one fact has been overlooked in a great deal of the discussion that has gone on concerning iron therapy in anemia:—the type of anemia studied. In many of the anemic conditions in which iron has been used, there is no reason why any form of iron, organic or inorganic, should be of any value whatever. The cause of the anemia is the gist of the question: there is a vast amount of difference between a nutritional anemia in an infant, presumably due to dietary deficiency, and an anemia in an adult due to carcinoma or chronic nephritis—and still they both are called "secondary anemias." If a patient is taking a diet containing enough iron and pigment with which to synthesize an adequate amount of hemoglobin, as he most certainly will be if he is taking an ordinary diet, there is no reason to suppose that the addition of any medicinal iron preparation should be of value, and as a matter of fact it is not likely to be. We believe that there are relatively few anemic conditions in which iron is of value (organic or inorganic), chlorosis and nutritional anemia due to iron deficiency being the two most outstanding; also possibly anemia after severe hemorrhage. We think it may be said, from a study of the literature, that the preponderance of evidence is in favor of the fact that inorganic iron can be absorbed and utilized in hemoglobin formation, when used in the right sort of case, and that it is probably more efficient if a pigment is also supplied.

Schmidt[6] fed adult white mice on an iron-poor diet of milk and rice. This did not produce anemia in them, but their offspring showed marked anemia, and did not grow as well as controls. The group of mice kept on the standard diet of milk and rice, with iron oxide added, gave birth to normal young.

McGowan and Crichton[7] duplicated almost the same investigation on pigs, with the same results.

Hart and his co-workers,[8] in a very careful piece of work on rabbits, arrived at the following conclusions:

(1) Rabbits limited to a whole (cow's) milk diet develop nutritional anemia.
(2) Inorganic iron ($Fe_2O_3$) added to the basal ration will not *per se* correct the anemia.
(3) Chlorophyll, free from iron, will, in presence of added inorganic iron, correct such an anemia as is induced by a milk diet.

Mitchell and Schmidt[9] did much the same work on mice. They found that egg-yolk, meat, and spinach, when added to the basal ration of milk, were efficient in correcting the anemia. They also used inorganic iron (Ferric chloride, ferric ammonium citrate, ferric oxide, and ferrous carbonate), and found that the first two, which are soluble, were very efficient in building hemoglobin; the last two, which are insoluble, were very inefficient. They suggest that the question of the solubility, rather than the organic or inorganic nature, of an iron compound may be the factor in determining its value in hemoglobin formation.

Whipple[10] and his associates, in many important and painstaking investigations, have studied the effect of various foods and iron compounds in hastening the regeneration of blood in dogs made anemic by repeated bleeding. They believed, in their earlier investigations, that inorganic iron was of no value, but in their later work, with longer periods of anemia, found it was of considerable value. Of the foods, liver was by far the most efficient in synthesizing hemoglobin, and they suggest that their work indicates that there is stored within the liver, parent pigment substances which may be easily utilized for hemoglobin formation.

The foregoing brief review of a small portion of the literature shows, in a general way, the methods of investigation employed, and while perhaps some of the experiments are artificial, and not to be applied to anemic infants, they serve as a general guide.

We have attempted to repeat the same sort of investigation in infants with nutritional anemia; that is, put them on a basal diet containing practically no iron, and very little pigment, and then added various iron preparations, and iron-containing foods in sufficient amounts to cover the iron and pigment needs, and observed the results. The infant with nutritional anemia, if other factors, such as infection, rickets, and digestive disturbances, can be excluded, is an ideal subject in which to study the effect of various foods and iron preparations in hemoglobin synthesis. It is extremely difficult, however, to secure ideal experimental conditions, and if these are not attained, the work is worthless. If a baby has severe rickets, or develops an infection of any sort, as babies so commonly do in the wards of any infants' hospital, additional factors are introduced which render that baby useless for purposes of investigation, and we were disappointed many times in this way, so that the series of cases which we were able to control, and in which we feel that the results are accurate, is not as large as we should wish.

The method of investigation was as follows: For infants under 9 months of age the basal diet was an ordinary milk modification with dextri-maltose or Karo, calculated according to the caloric needs. No cod-liver oil or orange juice was given. For older infants the basal diet was whole milk, about one quart daily, and farina. Both of these diets contain so little iron that it is negligible, and very little pigment. The hemoglobin determinations were made with a Newcomer hemoglobiometer (Bausch and Lomb), which we found very satisfactory, and

reasonably accurate. The food or iron preparation to be tested was added to the basal diet, and hemoglobin determinations made once a week. If no appreciable increase took place in two weeks, another factor was substituted or added. The iron preparation used was reduced iron, two grains twice a day. This was given in powdered form, shaken up in the milk. The liver was at first prepared as follows: finely ground raw liver was put into a small cheese-cloth bag, and this bag then dipped into boiling water for about two minutes. This slightly cooked liver was then made finer by mashing with a spoon, and was fed to the infant as a thick soup. More recently we have been using raw liver, ground so finely that it is in the form of a paste. This is mixed with the milk in the nursing-bottle, and readily goes through a fairly large nipple hole. From two to four tablespoonsful of liver a day were given.

No. 1. Age 2 mos.

| Date | Hgb. | Treatment |
|---|---|---|
| Dec. 16 | 54% | Reduced iron |
| Dec. 23 | 62 | " " |
| Jan. 7 | 60 | " " |

No. 2. Age 4½ mos. (Twin.)

| Date | Hgb. | Treatment |
|---|---|---|
| Dec. 17 | 52% | Reduced iron |
| Dec. 23 | 59 | " " |
| Dec. 30 | 50 | " " |
| Jan. 7 | 59 | " " |

No. 3. Age 19 mos.

| Date | Hgb. | Treatment |
|---|---|---|
| Dec. 19 | 41% | Egg yolk (1) |
| Dec. 27 | 30 | " " |
| Jan. 3 | 45 | " " (2) |
| Jan. 13 | 31 | " " |

No. 4. Age 8 mos.

| Date | Hgb. | Treatment |
|---|---|---|
| Feb. 6 | 31% | Reduced iron |
| Feb. 13 | 31 | " " |
| Feb. 20 | 36 | " " |
| Feb. 27 | 38 | " " |
| Mar. 5 | 44 | " " |

No. 5. Twin. 5 months.

| Date | Hgb. | Treatment |
|---|---|---|
| Nov. 7 | 63 | Ultraviolet Light |
| Nov. 14 | 62 | " " |
| Nov. 22 | 63 | Reduced iron (Light omitted) |
| Nov. 29 | 77 | " " |
| Dec. 3 | 79 | " " |

No. 6. Twin. 5 months.

| Date | Hgb. | Treatment |
|---|---|---|
| Nov. 7 | 64 | Egg yolk (1) |
| Nov. 14 | 62 | " " |
| Nov. 22 | 64 | Reduced iron (egg omitted) |
| Nov. 29 | 77 | " " |
| Dec. 3 | 84 | " " |

No. 7. Age 8 months.

| Date | Hgb. | Treatment |
|---|---|---|
| Feb. 6 | 27 | Ultraviolet Light |
| Feb. 13 | 34 | " " |
| Feb. 20 | 34 | Phyllosan (a chlorophyll preparation) + Light |
| Feb. 27 | 27 | " |

No. 8. Age 2½ years.

| Date | Hgb. | Treatment |
|---|---|---|
| Jan. 22 | 26 | Reduced iron |
| Feb. 7 | 40 | " " |

No. 9. Age 11 weeks.

| Date | Hgb. | Treatment |
|---|---|---|
| May 9 | 67 | Reduced iron |
| May 19 | 63 | " " |
| June 2 | 65 | " " |
| June 16 | 62 | " " |

No. 10. Age 20 months.

| Date | Hgb. | Treatment |
|---|---|---|
| July 21 | 32 | Spinach 4 tbsp. |
| July 28 | 36 | " |
| Aug. 12 | 32 | Spinach + reduced iron |
| Aug. 19 | 35 | " |

No. 11. Age 17 months.

| Date | Hgb. | Treatment |
|---|---|---|
| Mar. 4 | 36 | Mixed diet (egg, vegetables, soup) |
| Mar. 11 | 38 | Iron plus mixed diet |
| Mar. 18 | 36 | " |
| Mar. 25 | 43 | " |
| Apr. 1 | 44 | " |
| Apr. 8 | 45 | " |
| Apr. 16 | 44 | " |

No. 12. Age 2 7/12 years.

| Date | Hgb. | Treatment |
|---|---|---|
| Dec. 20 | 30 | Reduced iron |
| Dec. 31 | 51 | " " |
| Jan. 16 | 75 | " " |

No. 13. Age 17 months.

| Date | Hgb. | Treatment |
|---|---|---|
| Mar. 9 | 46 | Liver (2 tbsp.) |
| Mar. 18 | 55 | " |
| Mar. 25 | 57 | " |
| Apr. 1 | 59 | Iron added |
| Apr. 8 | 62 | " " |
| Apr. 16 | 61 | " " |

No. 14. Twin. Age 7 months.

| Date | Hgb. | Treatment |
|---|---|---|
| Feb. 11 | 21 | Reduced iron |
| Feb. 18 | 26 | " " |
| Feb. 24 | 26 | " " |
| Mar. 3 | 34 | " " |
| Mar. 10 | 31 | Liver—(omitted iron) |
| Mar. 19 | 47 | |
| Mar. 26 | 46 | |
| Apr. 2 | 51 | |
| June 8 | 80 | |

No. 15. Age 14 months.

| Date | Hgb. | Treatment |
|---|---|---|
| May 14 | 35 | Liver |
| May 21 | 33 | " |
| May 28 | 33 | " |
| June 4 | 33 | Liver + reduced iron |
| June 11 | 46 | |
| June 18 | 56 | |
| June 27 | 62 | |

No. 16. Age 3 4/12 years.

| Date | Hgb. | Treatment |
|---|---|---|
| Mar. 28 | 25 | Liver |
| Apr. 5 | 22 | " |
| Apr. 13 | 25 | " |
| Apr. 21 | 30 | " |
| Apr. 27 | 31 | Liver + iron |
| May 4 | 37 | " |
| May 11 | 51 | " |

Seen in July, and color appears perfectly normal. No opportunity to do Hgb.

CONCLUSIONS

1. Nutritional anemia in infancy is a deficiency disease, comparable to rickets and scurvy.

2. There is no reason for supposing that there is any active injury to the blood going on (i. e. hemolysis).

3. Inorganic iron alone (Ferrium reduction) will in some cases bring about a satisfactory rise in hemoglobin, indicating that inorganic iron can be used in hemoglobin synthesis.

4. Liver alone is also efficient in some cases, but the most rapid hemoglobin synthesis is effected by the use of liver and iron together.

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# ACUTE YELLOW ATROPHY: THE INCIDENCE OF THE DISEASE AND THE REPORT OF A CASE

BY HORACE K. SOWLES, M.D., F.A.C.S.*

ACUTE yellow atrophy of the liver is considered to be a rare disease and is extremely fatal. Its etiology is uncertain. The clinical picture may allow one to make the diagnosis with reasonable accuracy, but the final diagnosis must usually be confirmed by the pathologic findings in the liver itself.

The macroscopic findings in the later stages of the disease show a small liver which usually has an irregular, pebbled external surface. The cut surface is pale and often stained a bright yellow from the bile which has been liberated from the destroyed liver cells. There is some difference of opinion as to whether one may find an enlarged, swollen liver during the early process of the disease or whether true acute yellow atrophy is characterized from the beginning by a more or less rapid diminution of liver size.

Microscopically, there is gross destruction and necrosis of liver cells, often so extensive that over a considerable area no normal liver cells are found, because they have been completely destroyed or reduced to a mass of structureless fatty debris. Where the liver cells do persist, there is apt to be marked fatty degeneration. There is practically no evidence of any attempt at regeneration on the part of the liver cells, but often a very marked proliferation of bile ducts and early connective tissue.

*For record and address of author see "This Week's Issue," page 282.

The above pathology is definite but clinically there are apparently numerous possible etiological factors. Often one or more of these recognized causes may be demonstrable in a given case, but occasionally none of them can be found. Acute yellow atrophy seems sometimes to be the terminal stage or the direct sequela of other liver disorders, such as acute catarrhal jaundice or hepatitis.

It is well recognized that the disease is often closely associated with pregnancy and also with chemical poisons, such as chloroform, arsenic, and phosphorus. The hazard in regard to chloroform is chiefly when it is used as an anesthetic, and its dangers are possibly overestimated. In regard to arsenic, acute yellow atrophy is supposed to be one of the risks of treatment with salvarsan or arsphenamin, and there is an industrial hazard in the manufacture of some of the explosives in which phosphorus is used. Buechner points out that there is often in the early stages a very definite clinical difference between the yellow atrophy due to the above poisons and the classic acute yellow atrophy; the tendency in cases of poisoning being a more gradual onset with mild jaundice, a preliminary increase of liver volume due to fatty degeneration, less tendency to delirium, and infrequently leucin and tyrosin in the urine. In the later stages, however, the two conditions cannot be distinguished from each other.

Acute yellow atrophy is also supposed to be associated with syphilis, especially after treatment with arsphenamin, as just mentioned. In this instance it is not clear whether the toxins of

the treponemata have caused a lowered resistance of the liver cells or whether the treatment with arsenic is the more important factor. Herxheimer and other German writers have reported a marked increase in acute yellow atrophy in different districts of Germany during the last years of the war and the years immediately following the war, and raised the question as to the possibility of faulty nutrition as a cause. However, other writers attribute a great percentage of the increase during these years to poor or improperly combined salvarsan, which was on the market during that time, owing to a lack of proper raw materials for its manufacture.

According to Murowasky, as high as eighty per cent. of all syphilities treated with salvarsan were temporarily jaundiced and Herxheimer states that one-fifth of all published cases of acute yellow atrophy are luetic. This figure, however, may roughly correspond to the incidence of lues among certain groups of the population and in that case lues would be of no great importance in this consideration.

From all these factors one can readily assume that a specific poison or infection as a cause of acute yellow atrophy, is extremely unlikely and that it is really a symptom complex with a varied etiology, possibly the end stage of a toxic hepatitis in which the resistance of the liver cells has been so reduced or their susceptibility to disintergration so increased that complete autolysis takes place.

Therefore, it is not unreasonable to suppose that almost any severe intoxication or infection, particularly those which may continue over a considerable period of time, may so damage the liver cells as to cause the beginning of a process which may end in acute yellow atrophy. In fact, acute yellow atrophy of the liver has been encountered with erysipelas, cutaneous abscesses, septic gangrene, suppurative salpingitis, and osteomyelitis.

The clinical picture of acute yellow atrophy, according to Osler, presents three classical symptoms:

1. jaundice with vomiting
2. diminution of liver volume
3. leucin and tyrosin in the urine

Incidentally, as in most diseases, the picture is not always a perfect duplicate of the textbook description. For instance, pain is not supposed to be a dominant symptom and yet every case of a series reported by Buechner began with sudden pain in the upper abdomen. In half of them the jaundice appeared immediately with the nausea or within one to three days. In the other half the jaundice was delayed from seven to fourteen days. The diminution of liver volume is almost always present, but one may not be able to demonstrate it clinically until the disease is far advanced.

There are various other symptoms which are often helpful. The stools are practically always clay colored but they may give a positive chemical test for bile because if there is any obstruction in the common duct, it is merely incidental and not a part of the disease of acute yellow atrophy. The absence of bile in the stools is not due to obstruction but to lack of secretion. The urine on the other hand contains large amounts of bile as the result of liver destruction.

There are almost invariably mental changes, characterized either by delirium or drowsiness. Sometimes this symptom is present from the early stages, but in other cases does not appear until late in the disease. The blood, except for a possible tendency toward abnormal bleeding, is not remarkable. A marked diminution of urine output is usually present and often, nearly complete anuria in the later stages of the disease.

Rarely there is a case which might be classified as chronic yellow atrophy or perhaps as an arrested yellow atrophy. The writer saw one such case in whom the diagnosis of acute yellow atrophy was made at operation and confirmed by pathological examination of a specimen from the liver in 1926. This patient was alive and well in 1928 but unfortunately cannot be traced at the present time to check up in regard to present physical condition and present liver function. This case would seem to indicate that in some individuals the destructive process becomes arrested and regeneration of the liver cells takes place. Probably this happens much more often than we realize and, because the patient recovers, the diagnosis of yellow atrophy is never made.

In reviewing all the records of the Massachusetts General Hospital up to 1927, the diagnosis of acute yellow atrophy appears twenty-five times. The first case was in 1894 after which the diagnosis did not appear again until 1908 when there were two cases. From 1914 to 1922 there was about one case each year and fifteen cases from 1922 to 1927.

Only one of these twenty-five cases was discharged from the hospital alive. This was a young married woman of twenty-three who was discharged unrelieved in 1915 with a diagnosis of probable acute yellow atrophy of the liver. This patient became jaundiced three weeks after delivery from a normal pregnancy. The jaundice at the time of admission had been present for seven weeks. She stayed in the hospital three weeks. Clinically this patient showed enlargement of the liver but no leucin or tyrosin in the urine. The diagnosis in this case must be considered as not proven and there was no follow-up record.

Out of the twenty-four fatal cases, a postmortem examination was made in eighteen and in seventeen instances a positive diagnosis of acute yellow atrophy was confirmed by a microscopic examination of the liver. One case was classified pathologically as an atypical cirrhosis of the liver. Of the six fatal cases in which no autopsy was done, three were typical enough to be classified as acute yellow atrophy with reasonable certainty. In the other three

cases there were factors which might cause one to have some doubt as to the accuracy of the diagnosis. A very brief summary of these six cases is here given.

1. A male, aged 47, admitted in 1926. Jaundice of two weeks' duration. Leucin and tyrosin in the urine. A typical clinical picture. No apparent etiology. Probably a real acute yellow atrophy.
2. A female, single, aged 39, admitted in 1926. A typical clinical picture of jaundice and vomiting with slight pain and chills, very rapidly progressive, and resulting in death in five days. Probably a real acute yellow atrophy.
3. A female, widow, aged 45, admitted in 1926. Jaundiced for three weeks. No apparent cause. A definite clinical picture which showed a progressive shrinkage in the size of the liver. No leucin or tyrosin in the urine. Probably a real acute yellow atrophy.
4. A female, married, aged 30, admitted in 1926. She had a septic abortion three weeks previously. She had been mildly jaundiced for eight days and came in with a very high temperature, high white blood count, extremely toxic, probably a septicemia. Death in this case may very well have been due to sepsis with secondary hepatitis rather than a real acute yellow atrophy.
5. A male, aged 31, admitted in 1916. Diagnosis was made of acute syphilis, alcoholism, delirium tremens, nephritis, and a question of acute yellow atrophy. There was no leucin or tyrosin in the urine. The patient became jaundiced next day after treatment with salvarsan and died four days later. There may well be reasonable doubt about the accuracy of acute yellow atrophy in this case.
6. A female, married, aged 34, admitted in 1922. The patient was known to have active syphilis. She had been jaundiced for two weeks before admission, following treatment with salvarsan. A definite question of acute arsenical poisoning was raised at that time. Probably this case was not a real acute yellow atrophy.

From a summary of these statistics we, therefore, have twenty cases of undoubted acute yellow atrophy in the Massachusetts General Hospital from 1894 to 1927. As a possible etiological factor in these cases, we have pregnancy in one, syphilis or treatment with salvarsan in three cases, a patent medicine which was supposed to contain arsenic in one case. In the remaining fifteen cases, no positive etiological factor can be determined from the history. During these same years, from 1894 to 1927, there were approximately 200,000 admissions to the Massachusetts General Hospital so that the proportion of acute yellow atrophy to admissions is one in ten thousand. Therefore, I think we can justly classify it as a rare disease. However, the number seems to be increasing in more recent years.

Finally I wish to report a recent case of acute yellow atrophy in which the symptoms were somewhat complicated and the diagnosis obscured by a large ovarian cyst.

Mrs. J. C., a widow, aged 64, was sent into the Faulkner Hospital by Dr. Howard Jackson, December 3, 1928. The patient complained of pain, jaundice, and abdominal distention.

For two months previous to admission the patient had been having increasing difficulty with constipation. Before that time her bowels had been regular and normal. One week before admission, patient became jaundiced and had some abdominal pain of a cramplike nature more or less typical of pain due to gall bladder disease. For the last two days this pain had been quite troublesome and there had been some vomiting with considerable distention. Very little result had been obtained from enemata and cathartics. Physical examination showed a patient who was apparently in good physical condition but deeply jaundiced. She had rather marked abdominal distention so that one could feel nothing in the abdomen. It was said that some days before it had been possible to feel an indefinite mass in the lower abdomen. There was slight tenderness over the right upper quadrant but the area of liver dullness was not increased.

It was thought that on account of the rather marked changes in bowel habits during the last few months, the present distention, and a question of a partial obstruction, she might have a neoplasm of the large intestine with metastases to the liver. Possibility of primary malignant disease of the bile ducts or metastatic interference with the bile ducts, or simply cholelithiasis and cholecystitis were considered and exploration advised.

Under ether anesthesia incision was made in the right upper quadrant over the gall bladder region. There was some bile tinged fluid in the abdominal cavity. There was a large mass in the lower abdomen. The incision was extended downward a little and a cyst of the right ovary as large as a football was delivered. It had a small pedicle which was easily tied and cut and the cyst removed. There was another cyst three or four inches in diameter firmly adherent in the pelvis and numerous small calcified fibroids in the uterus.

In view of the findings in the liver, it was thought best to do nothing further in the pelvis. There was no sign of any inflammatory reaction around the gall bladder which was normal in appearance, soft, and compressible. The liver was very small with a rough, pebbled surface. The diagnosis of acute yellow atrophy was made and the abdomen closed. The patient made a good ether recovery and the next morning said that her abdomen felt much better. Evidently the removal of the large cyst had relieved some of the intra-abdominal tension. That afternoon, however, she became rather drowsy and at times irrational. The next day she developed a definite coma and died on the third day after operation. The post-mortem examination of the abdominal cavity showed that the liver had diminished very markedly in size since the operation three days before. A specimen of liver was removed for pathological examination and showed the typical picture of acute yellow atrophy.

The pathological report, of Dr. J. S. Rooney, was as follows:

"Specimen consists of a small piece of liver 3.5 by 2.5 by 1 cm. It is almost yellow in color and firm but friable.

Sections microscopically through the liver tissue show areas where the liver cells are wanting and the area is being replaced by newly formed connective tissue. There is a considerable quantity of necrotic debris in these areas. There is marked proliferation of the bile ducts in many places. In other areas of the tissue hemorrhages are noted and the liver cells contain many fat vacuoles. The histological picture is one commonly described as acute yellow atrophy.

Diagnosis: Acute yellow atrophy of the liver."

In summary I would say that acute yellow atrophy of the liver is a rather unusual and extremely serious disease of varied toxic etiology. The differential diagnosis between it and the other causes of jaundice is often uncertain and in fact some of the other causes of jaundice may develop into acute yellow atrophy. The treatment is unsatisfactory and of little avail. In general, fluids, glucose, alkalies, and relief of

pain and vomiting are indicated. Exploratory operation is usually done and is justified because of the chance that one might overlook a condition curable by surgery. However, if we have a positive diagnosis of acute yellow atrophy, operation is not indicated.

## MEDULLARY CARCINOMA OF THE OVARY IN A GIRL TWELVE YEARS OLD

BY JOEL ROSENBERG, M.D.*

I believe that an understanding of the condition indicated by the title of this paper will be clarified by a short review of the embryology of the ovary.

*EMBRYOLOGY.* The first sign of development of the reproductive gland is a heaping up of epithelium on the urogenital ridge. In the female, the Wolffian ducts degenerate and the genital ridges produce ovaries. The mass of tubules extending between the ovary and tube is recognized as the epoöphoron. A medial and a lateral fold which surround the gland are known as the mesovarium.

The reproductive organ, consisting of an epithelial nucleus, is divided from its overlying superficial epithelium by a layer of connective tissue, known as tunica albuginea. This layer prevents the superficial epithelium from taking any part in the formation of ova. Blood vessels and connective tissue soon begin to invade the epithelial nucleus; also the formation of a coarse medullary and fine cortical zone is noted, and the transformation of cells into young ova. Young ova degenerate at the center and in this destructive process the meshes are filled in with connective tissue, thus forming the stroma of the adult ovary. Each ovum is surrounded by a layer of cells, forming the primary follicle. Both ovaries lie above the brim of the pelvis and sink into the true pelvis after the first year of life. The ovary assumes its adult characteristics in the third year.

*ORIGIN OF TUMORS.* There are numerous theories as to the origin of ovarian tumors. Von Recklinghausen, as reported by Taylor[1], believes that all tissue of extra-ovarian origin is largely responsible for tumor growths of the ovary. Goodall[2] states that the tubules of the Wolffian body never invade ovarian tissue. Sampson[3] developed the theory that tumors of the ovary have their origin in endometrial implants.

*AGE AND INCIDENCE.* Solid carcinoma of the ovary occurs most frequently during sexual activity; as stated by Chenall[4], between the ages of twenty-five and forty-seven. The type of tumor most commonly found in the young is the sarcoma, but many authors have reported cases of solid carcinoma in children as young as eight years old. The youngest was reported by Wiel[5], that of Chiene, a child three months old.

Leopold, according to Briggs and Walker[6], was the first to publish a series of fifty-nine solid tumors of the ovary. Gussenbauer[7] and Leopold each reported a case, eight and nine years old respectively.

In an analysis by Kelly[8] of 126 cases of tumor of the ovary in children, 24, or 19%, were solid; of the solid type 5, or 20%, were carcinoma, or about 3.9% of all tumors were carcinoma.

*CLINICAL SIGNS AND SYMPTOMS COMMONLY FOUND IN CHILDREN.* The child is first noted to be less active and is listless, although she may not complain at this time, except perhaps of disturbances in micturition and defecation. Early in the disease abdominal enlargement is noted with a gradually failing appetite. Menstrual disturbances may occur but are not always present. Fatal termination in children seems to be quite rapid, death occurring two to nine months after the onset of the first smptom, if operation is not performed early.

*TREATMENT.* It is generally conceded that early operation is by far the treatment of choice. The type of operation is, however, left to the individual surgeon. Some advise the removal of both ovaries, if one is involved, regardless of the age of the patient. Others believe that removal of the affected ovary will result in a cure. Spencer[9] states that if tumors are small and retain more or less the shape of the normal ovary and have a distinct capsule, the affected ovary alone may be removed with safety. Frequently the tumors occur bilaterally due to the fact that when fetal rests occur in one of the bilateral organs, they usually occur in the other.

*REPORT OF CASE.* C. G. A female child, 12 years old, entered the Beth Israel Hospital, October 24, 1928. She complained of weakness and of tiring very easily. The mother said the child had refused to play, had become listless and lost weight, and she had noticed that the child was growing paler. Although the mother told that the onset of the present illness was only of two weeks' duration, she admitted that the child had become very constipated for the past four months, and had noted occasional nocturia.

*MENSTRUAL HISTORY*: Catamenia began six months before entrance. No dysmenorrhea; always regular. No abnormal discharge.

*PAST HISTORY*: Negative except for frequent attacks of sore throat, for which she had her tonsils removed six years ago.

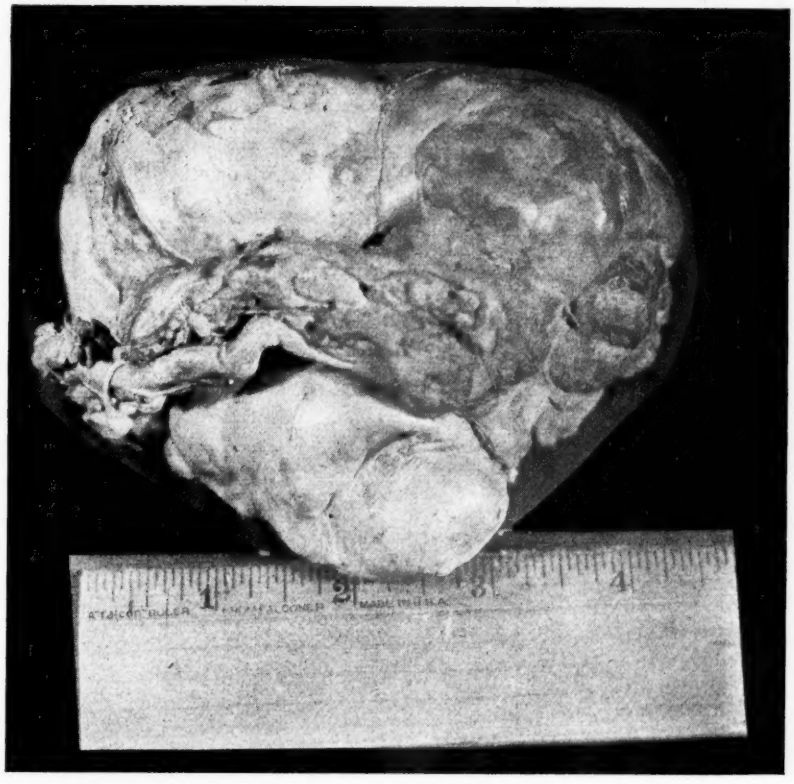
*CIRCULATORY AND NEUROMUSCULAR SYSTEMS*: Negative.

*GASTRO-INTESTINAL SYSTEM*: Negative except for a failing appetite.

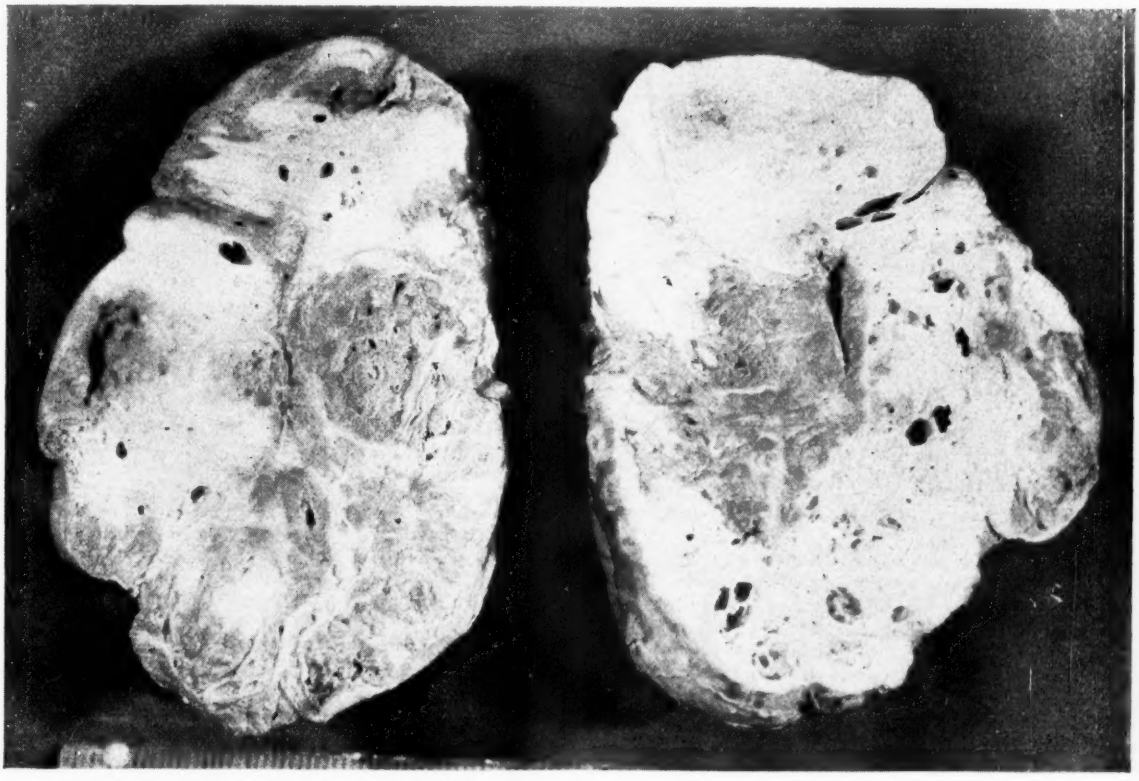
*FAMILY HISTORY*: Paternal grandfather had diabetes; otherwise negative.

*PHYSICAL EXAMINATION*, revealed a well nourished and well developed young female, apparently

*For record and address of author see "This Week's Issue," page 282.



1. SURFACE VIEW OF TUMOR. Showing encapsulation, lobulation, and complete obliteration of ovary. The Fallopian tube is firmly attached. The severed pedicle is at the right end of the tube.

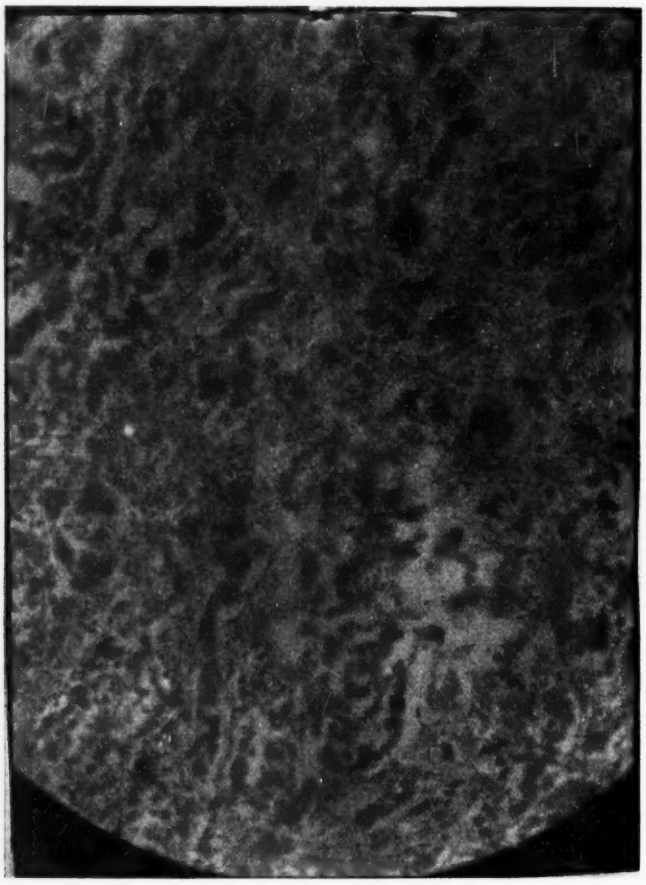


2. LONGITUDINAL SECTION OF TUMOR. Showing the lobulated structure and areas of cystic degeneration. The darker areas represent hemorrhagic necrosis

comfortable. Lungs negative. *Heart*—loud blowing systolic murmur heard all over the precordia but especially at the apex. No enlargement. B. P. 110/70. Breasts not enlarged.

The abdomen presented a hard, irregular, nodular mass, freely movable, rising from the pelvis to one inch below the umbilicus. This mass was not particularly tender and lay directly in the midline. There was some mucoid vaginal discharge. The rectal examination was essentially negative except for pressure from the overlying mass.

*PATHOLOGICAL REPORT.* The specimen consists of one large, well-encapsulated mass of tumor, the whole body of the ovary being involved rather in uniform growth throughout. Through the peritoneal coat, which is now stretched and thinned by the underlying tumor tissue, is disclosed an irregular nodular surface beneath it. The color as seen through this capsule is a mixture of alternate greyish and yellowish discoloration. These appear as separate blotches, and in various combinations of the two, with neither color predominating. The tube



3. LOW POWER PHOTOMICROGRAPH. Showing a rapidly growing area of medullary type, with also considerable lymphoid cell infiltration.

X-ray of kidneys, ureters and bladder reported as negative, but a dense shadow corresponding to the mass was noted.

The urine on two examinations was negative.

*BLOOD EXAMINATION*: R.B.C. 4,700,000; Hbg. 80; W.B.C. 7,000; smear negative, platelets negative.

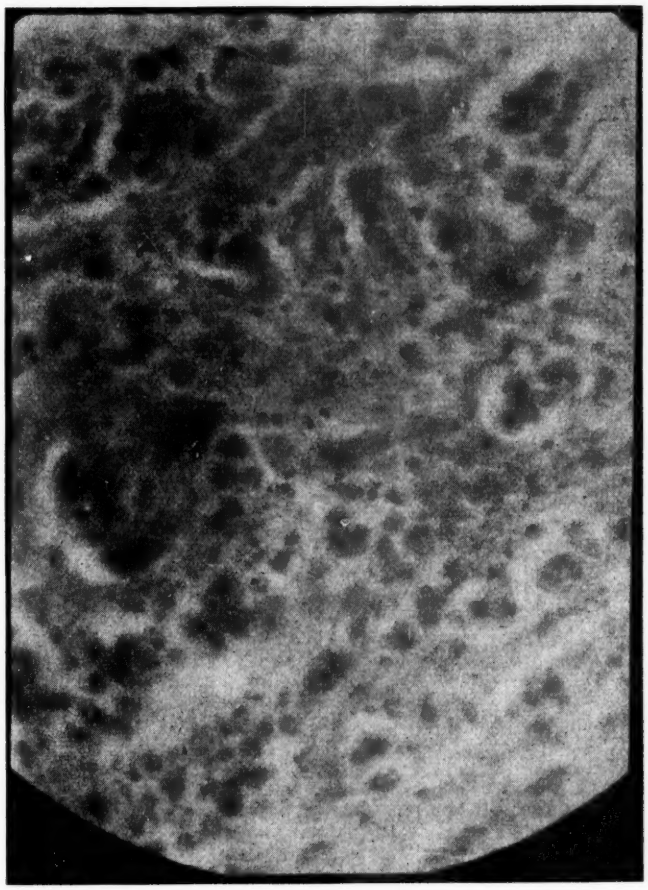
*OPERATION*: Four days after admission, operation under general anaesthesia was performed by Dr. Wyman Whittemore. A large tumor of the left ovary together with the left tube was removed. This hard grayish white mass was found filling the pelvis behind the uterus and was adherent to the omentum and intestines. The right ovary and right tube appeared normal and were left untouched. The patient made an uneventful convalescence, the wound healing well and she was discharged on the fourteenth post-operative day.

The patient was referred on discharge to the x-ray department for deep x-ray therapy and when last heard of, six months later, her menstrual periods had ceased and she was attending school free from symptoms, happy and doing well, with no clinical evidence of recurrence.

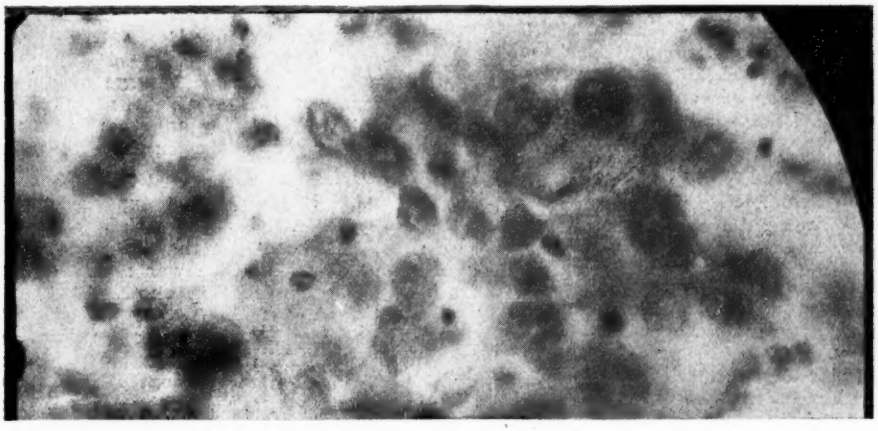
is still adherent. It measures 10 x 0.5 cm. in size. The surface of the tube suggests a somewhat mild congestion. The tumorous ovary weighs 520 grams, and is egg-shaped. Measurements cannot be ascertained accurately, due to its irregular appearance, but approximately it is 14 x 10 cm. in cross diameters. On dividing the entire tumor mass along its longitudinal axis, the blood supply is found to be fairly good. However, the oozing may not be due entirely to its vascularity, but perhaps to small hemorrhagic cystlike inclusions. As the tissue is examined from the periphery toward the center, there are fairly large, cheesy, necrotic areas encountered, which vary in the intensity of their destructive tendencies, and which reach a most advanced degree at about the center. These necrotic areas are broken down, and their contents feel sandlike on handling. Other spots adjoining these portions appear as small pigmented nests. This is, in all probability, the remains of degenerated and transformed blood into resulting pigmented material. The whole mass may be considered rather firm, and there is, aside from the pigmentation, a uniform pinkish-white tinge throughout the

tumor mass. Some of the crevices which are found around the sides of the lobulated masses that extend through the surface, pass deeply into the stroma. The demarcation is made up, therefore, of a thin band of fibrous tissue, which appears grossly as a faint line. Each lobule seems to have this same characteristic, which perhaps shows that each at one time possessed an individual capsular structure.

Microscopic section shows large masses of epithelial cells separated by broad bands of fibrous tissue septa. In the center of the masses the stroma is very delicate, consisting of thin strands of fibrous tissue, dividing the tumor cells into small packets. Within these packets the cells are very loosely arranged, and in no place are there any large groups or columns of closely applied cells. As the septa



4. HIGH POWER PHOTOMICROGRAPH. Showing the tendency to grow as isolated, separated cells.



5. OIL IMMERSION PHOTOMICROGRAPH. Show ing marked irregularity of the hyperchromatic nuclei.

are approached, the stroma becomes more dense, and here the groups of tumor cells are surrounded by considerable fibrous tissue. Nowhere is there any tendency for these epithelial cells to line these spaces or to form alveoli. Even within the septae scattered rows of tumor cells are found between the meshes of the fibrous tissue. Large areas are necrotic, but still show a shadow picture of the original structure, resembling, in this way, recent infarcts. They usually extend to a septum, the tissue on the opposite side of the septum being still viable. Within these necrotic areas one often sees a large blood vessel with viable tissue about it, and from this, young fibroblasts growing out to organize the necrotic area. In the better preserved areas the tumor is seen to be made up of small hyperchromatic vesicular nuclei, often showing one or two nucleoli, and a very definite nuclear membrane. Most of the nuclei are fairly uniform in size and shape, being round or slightly irregular. In some areas there is considerable variation in the size of the nuclei, but no cells with very large nuclei are found. Mitotic figures are present in moderate numbers, an average of one per oil immersion field being found in some places. The cytoplasm of these cells is rather scant in amount, being present as a thin, irregular rim about most of the nuclei. It stains purplish and is very granular. Most of the cell borders are easily made out. Multinuclear tumor giant cells containing two to four nuclei are found in small numbers. The tumor cells seem to have a very loose relation to one another, for even in the largest groups found, the cells can easily be made out separately. Intermingled with the tumor cells throughout are degenerated cells with pyknotic nuclei and acidophilic cytoplasm. There is also a rich sprinkling of lymphocytes throughout the tumor.

The general picture is that of a rapidly growing epithelial tumor, presenting very little evidence of differentiation of any type.

I am deeply indebted to Dr. William S. Cooper, of the Department of Pathology, for the invaluable aid he has rendered.

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## NOTES ON SOME EYE ACCIDENTS OF JULY 4th*

BY WALTER C. CAMERON, M.D.†

A brief report of the serious eye accidents which occurred on the Fourth of July and were admitted to the wards of the Massachusetts Eye and Ear Infirmary appears to be of sufficient interest to warrant publication.

CASE I. In the exuberance of youth, E. D., age 11, opened a bottle of home brew by knocking the neck against a stone, exploding the bottle and driving glass against his left eye. Examination showed a perforating wound of the sclera, the lens being uninjured. No glass could be demonstrated in the eye. The wound was sutured, healing by first intention. The prognosis seems good.

CASE II. McG., aged 22, was struck in the left eye by a fragrant of a torpedo which was thrown at the car in which he was riding. He sustained a lacerated wound of the cornea, a prolapsed iris and a traumatic cataract. No intraocular foreign body could be demonstrated by x-ray. The iris was excised, the wound covered by a flap of conjunctiva and normal healing took place. At a later time the cataract will be removed and a certain degree of vision should be obtained.

CASE III. T., aged 33, got up in the night to observe a fire in the house next to him. Unfortunately for him a fireman turned the hose on the window through which the observation took place, shattering the glass and wounding the right eye. The consequences were a perforating wound of the cornea, prolapse of iris and traumatic cataract. No glass could be demonstrated in the eye. As in the previous case the iris was excised, and the wound covered with a conjunctival flap. Fairly satisfactory healing ensued. The cataract will have to be removed later.

CASE IV. C. H., a boy of ten, was watching his brother through the glass of the front door. In an excess of enthusiasm the brother's fist went through the glass, injuring the patient's right eye. There was a clean corneal wound with no prolapse of iris and it was treated expectantly under aseptic precautions. On the fifth day pain and increased pressure led to operation which consisted of incision of the cornea and removal of as much softened lens material as possible. At the operation a small sliver of glass was found embedded in the lens substance. It was washed out of the eye and the patient's progress is now satisfactory.

CASE V. E. M., male aged 6. The patient put a firecracker in a wooden spool, stuck the spool on a stick about three feet long and holding the stick in his hand, lit the cracker. With the explosion something struck his right eye. This was another lacerated wound of the cornea with no iris prolapse and no foreign body in the eye. As the apposition was good it was treated expectantly under aseptic precautions. This eye developed a mild infection and five days later after discharge became markedly inflamed and painful. With nil vision, enucleation was deemed advisable and the patient is undergoing a normal convalescence in the hospital at present.

CASE VI. McF., male aged 21. While walking along the street a firecracker exploded and the patient felt a sudden pain in the right eye. Unable to see with it he entered the hospital 1½ hours later. Vision in the left eye was normal, in the right eye was nil. Examination showed a gaping laceration of the cornea from 9 to 3 o'clock extending back through the ciliary body and sclera on each side abut 7 m.m. Vitreous, blood clot and a mass about 3/4 cm in diameter presented from the wound. The mass resisted gentle pulling and was not removed. The globe was soft to gentle palpation.

Enucleation with implantation of a glass ball was done after consultation. Pathological examination revealed the foreign body to be a section of a firecracker, one end of which touched the retina, the other presenting through the wound.

The patient had an uneventful convalescence with discharge on the seventh day after operation.

*From the Harvard Medical School Service—Massachusetts Eye and Ear Infirmary.
These cases are reported from the service of Dr. G. S. Derby.

†For record and address of author see "This Week's Issue," page 282.

CASE VII. C. D., aged 15. Thinking that he would start the Fourth off with a bang and also give his family a surprise C. D. arose at 5 and set off his toy cannon. The back fire through the fuse hole peppered the boy's forehead, eyelids, nose, cheeks and both eyes with multiple powder burns.

The corneal powder burns were removed in the clinic. He was found to be a diabetic and so had to have preoperative treatment before scrubbing of the face could be done, which was performed 24 hours later under ether.

Discharged in six days, the corneae were healed and vision was normal. The face still bore some powder burns which will have to be removed later.

Of the first six cases, it is possible that Case I will recover with fairly normal vision. In two cases the eye was irretrievably damaged and had to be removed. The other three suffered traumatic cataract, a serious injury. Although a definite amount of sight is likely to be retained in all, all must carry on their occupations as one eyed individuals, though having a second eye in reserve which may be brought into use should anything subsequently happen to the uninjured member.

## CERTIFIED MILK IN HONOLULU

BY FRANKLIN G. BALCH, M.D., F.A.C.S.*

IT was a great surprise to me, during a recent stay in Honolulu, to find out how much ahead of us they are in those faraway islands in the matter of having the public educated on the subject of certified milk.

They have certain advantages over us in the East in the matter of climate. It is so warm that the cows can always be out of doors. During February, which is their coldest month, I never saw the thermometer below 58 and that only for a short time in the morning. On the other hand I saw it at 82 and they say that is about as high as it ever goes.

Cows are not turned out to pasture, in the two certified farms on the island of Oahu, but live in large paddocks underneath the trees. All of the green stuff and hay is fed to them in great racks in the dry lots while the grain ration is fed under cover while they are being washed. Ten crops of alfalfa can be grown in a year, so there is no shortage of green food. On the other hand, the hay is brought from the coast and is a very necessary part of the ration. The prevailing trade winds blow pretty much all the time and carry away all dust and most of the flies. Both the Hind-Clarke dairy and the Waialae ranch are on the lee side of the islands, very near the shore but there is no collection of dust to be blown across the dairies. As I said, most of the flies are kept off by the constant breeze, but in addition there is a small ant which feeds on the larvae, so that flies are much less of a problem with them than they are with us. I saw almost none about any of the barns; there were a few in the calf barns the first day I went over the place, but the second day there were almost none; I saw none about the bottling room.

Two teams are kept busy most of the time carting muck out of the paddocks and carrying it off down onto the fields.

Another advantage, due to the climate, is that there are no closed barns. The buildings have stanchions and tight floors, just as we have in our best farms, but no sides. The cows come in from the dry lots down a narrow lane and as they come in, they walk through a shallow puddle of some creosote composition. They then go into the stanchions, where they are fed, and while being fed are washed. The washing process is quite different from ours. The dairy has its own artesian well and the cows are washed down with a hose. The water comes from the well at 70. The Japanese dairymen start in with a scrubbing brush and a hose and scrub and wash the cows all over. Their feet are carefully scrubbed off with a brush so that they are pretty nearly immaculate by the time they leave the stanchions and go into the milking stands beyond. Milking is done by machine. All the usual tests of foremilk, etc., are done first. From the milking rooms the cows go straight back to the paddocks.

All the cooling and bottling processes are the same as ours. They milk at twelve noon and twelve midnight. They say they have to milk at midnight so as to make delivery of that milking in time for breakfast, for people in Honolulu get up early.

I have mentioned only such deviations as there are from our customs in this particular part of the country, deviations due largely to the climate.

At the Waialae ranch they have a Jersey herd. At the Hind-Clarke dairy they have a Guernsey herd and a Holstein herd. The Hind-Clarke people have a large ranch over on Hawaii where they raise beef for the Honolulu market. They save all heifer calves from their dairy herd and raise them on the Holstein milk. They think this is better than the Guernsey milk for that purpose. They keep them at the dairy until they are five months old, when they are sent across to the ranch at Hawaii. The cows live there until after their first calf, when they are sent back to the dairy. There has never been a reactor in any of their cattle sent from the ranch. There are very few reactors anyhow, as there is less than two and one-half per cent. of tuberculous cattle on the whole island of Oahu. In the last test of the Hind-Clarke dairy, 409 animals were examined without a reactor.

*For record and address of author see "This Week's Issue," page 282.

When it is necessary to buy cattle outside of those coming from their own ranch on the "big island", as they call it, they put them in quarantine until the veterinarian is satisfied that they are perfectly safe. This veterinarian is appointed by the Medical Milk Commission. A test is made twice a year by the territorial veterinarian for tuberculosis.

And now comes the interesting part of the story. The Hind-Clarke dairy produces about 3900 quarts of certified milk a day. The Waialae dairy belongs to the Territorial Hotel Corporation and supplies some of the biggest hotels. The surplus goes to the Honolulu Dairymen's Association. They have about 100 cows with about 80 milking. About 700 quarts were being produced when I was at the island. Neither of these dairies produces anything but a certified milk. The Honolulu Dairymen's Association put out about 15,000 quarts and New Fair Dairy about 2000 quarts. These are pasteurized. I was told that about 2000 or 3000 quarts more are sold in Honolulu, probably all unpasteurized. That makes 20,000 quarts apart from the certified milk, or 4600 quarts of certified milk out of a total of 24,600. These figures are approximate.

The Guernsey milk, which is about 4.4 to 4.5 butter fat, is sold for thirty cents a quart, and the Holstein, to which about one-third Guernsey milk is added in order to bring it up to a butter fat content of about 3.7, for twenty-two and a half cents. Apparently it is the public who have become convinced that a certified milk is well worth the difference in price, for I think the doctors, as a whole, seem little more interested in the milk problem than our doctors are. In fact, the manager of the Hind-Clarke dairy told me that he had a lunch out at the farm, one day, to try to get the doctors to come and see what they were doing in the production of clean milk. He said they came, ate the lunch, and went straight home, for the most part. A few were interested, stayed, and went over the plant.

Last year the farm made fifteen per cent. on its investment.

Samples for examination are taken by the Board of Health ten times a month. They pay for the bottle, get it from the wagon and put it directly into a box full of cracked ice in the presence of the driver. They have to sign a receipt for the sample that they have bought. In this way the dairy is assured of a fair count and it seems as if it were the only proper way to take a sample for examination. The counts run for the most part from 1000 to 1500. Anyone can sell raw milk in Honolulu if the count runs under 50,000. Formerly the Hind-Clarke dairy used to do its own counts, as well as the Board of Health, but now they rely entirely upon the Board of Health.

It looks as if Boston, with its fraction of one per cent. of the total supply of milk from certified herds, is far behind Honolulu when that island can show a consumption of certified milk equal to about twenty per cent. of the daily supply.

People in Honolulu are thoroughly aroused to the desirability of clean milk. The supply for the big Punahou private school comes from their own farm. I went out there and inspected that dairy also. They are nearly as painstaking as the certified farms, though they do not have the doctor's inspection, etc. All their cows are tuberculin tested twice a year and everything about the milking house and the milking stanchions is as clean as can be. Their counts run very low. The milk is not pasteurized.

## A DOUBLE ADJUSTABLE KNIFE FOR CUTTING FASCIAL STRIPS

BY EDWARD M. HODGKINS, M.D., F.A.C.S.*

THE difficulties ordinarily encountered in procuring proper fascial strips for use as living sutures in herniorrhaphy are enough to discourage the ordinary operator, and may lead him to discard the procedure as impractical. My experience with this comparatively new method[1] has convinced me that successful suturing depends largely on the measured and uniform width of the strips, and that poorly prepared strips usually cause trouble. The technique most commonly used involves making two parallel incisions and tearing the strip away from its attachments; this is unsatisfactory, as the strip obtained is likely to be uneven in width, with the edges frayed and the body traumatized. Such a fascial suture frequently breaks off at the narrow points, and is difficult to draw through the tissues. Even if one is successful in weaving a poor suture into place, one can have little confidence in its ability at first to withstand muscular strain and later to organize in the tissues as a permanent tendinous band. I am convinced that the method will gain more credit if fascial sutures are accurately prepared, with careful attention to their width as applied to the individual requirements of each case.

The instrument here illustrated is an adjustable double knife designed to make two parallel incisions. The construction requires little de-

*For record and address of author see "This Week's Issue," page 282.

scription. Two Bard-Parker handle-tips are fused on opposite screw-threads so that, by means of a thumb-screw adjustment, the two parallel blades can be set anywhere from 1/12 up to 3/4 of an inch apart. It is possible thus to cut even fascial strips of proper width for any reasonable suture requirement. This knife was manufactured for me by Codman & Shurtleff, of Boston.

### TECHNIC OF USING KNIFE

The handle is grasped with the index finger extended onto the rest. The incisions are first started by moving the blades backward and forward by gentle pressure, until both blades have cut through the full thickness of the fascia. The knife is then held at an angle of approximately fifteen degrees to the surface of the fascia, so that the belly of the blades, not the points, will do the cutting. By drawing the knife slowly and exerting even pressure, a uniform and untraumatized strip is obtained.

While this knife is intended primarily for fascial work, it will prove valuable in other operative procedures whenever parallel incisions are desired. In excision of scar tissue and preparation of pedicled skin-grafts, for example, the grafts will always fit accurately into the bed of the excised scar if the adjustment of the blades is maintained the same for both operations.

I have now used this instrument extensively, and feel that I can recommend it to the surgeons who are interested in living-suture methods or pedicled-grafting work.

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## A CURE FOR CANCER IN 1720

BY ROBERT FRANCIS SEYBOLT*

TWO centuries ago, cancer research was quite unnecessary. A "Doctor Sharp of London" had discovered a cure which did not involve the "cruel practice of manual Operation." He was so sure of his treatment that he promised to waive the fee "if it should happen that he miscarries of Curing." On the occasion of a visit to this country, he published the following notice in the *Boston Gazette*, Oct. 3-10, 10-17, 24-31, 31-Nov. 7, 7-14, 1720:

"Doctor Sharp of London, being arrived at Boston on his return from Jamaica to England, and intending to stay but a few weeks in this Country, gives notice that he is to be advised with at Madam Leblonds in Tree-Mount Street, by those troubled with Cancered Breasts, other Cancerous, or Scrophulous Tumours, whether in the Throat or any part of the Body, the Kings Evil, Leprosie, Scurvy, Rheumatisms, or Stinking, Rotten Ulcers, proceeding from what Cause soever. And as he is a Stranger, to prevent the Calumny of designing, or ill-natured Men, promises, if it should happen that he miscarries of Curing, to take no Money.

N. B. He gives advice to the Poor gratis."

Four months later, he appeared "at Piscataqua, in the Province of New-Hampshire." He had either cured all the cancers in Boston, or had failed to find the encouragement which he desired. At this time, he announced (*Boston Gazette*, Mar. 6-13, 1721) that he was preparing for publication a comprehensive treatise on the subject:

"Shortly will be Published, A Treatise of Cancers: Wherein the Difference, Nature, Cause, Signs, and method of their Cure will be justly handled; and that common, but cruel practice of manual Operation is Exploded; in which the Author will demonstrate that it is not only Injudicious, but Desperate and Dangerous; directly opposite to those Sanative and Gradual Proceedings necessary to eradicate such Avoracious Disease: and that it is not only possible but probable to prevent its eating and spreading any farther upon the very first Application: which is obvious from Experience, Reason and Authority. With some Medicinal Observations and Prescriptions. Designed purely for the benefit of the People in New-England. By Dr. Sharp of London, now residing at Madam Story's, at Piscataqua, in the Province of New-Hampshire; where any may be cured of cancers, Kings Evils, Leprosies, or any Scrophulous Tumours or Ulcers.

N. B. Advice to the Poor gratis."

Sharp's "Treatise of Cancers" is not listed in the standard bibliographies of early eighteenth century publications. If it could be located, it would undoubtedly provide diversion for those who are engaged in a scientific study of cancer and its treatment.

*For record and address of author see "This Week's Issue," page 282.

# Case Records of the Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY R. C. CABOT, M.D.

F. M. PAINTER, A.B., ASSISTANT EDITOR

## CASE 15321

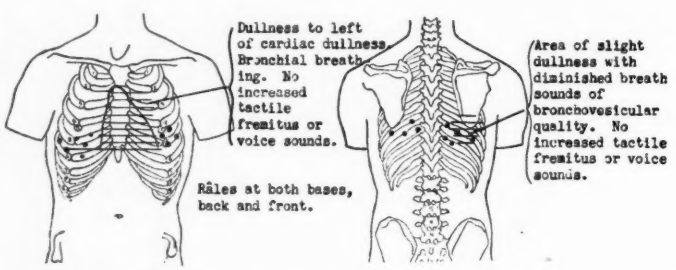
## SUDDEN DEATH WITH CARDIAC DISTRESS

An Italian of seventy-eight was sent to the Emergency Ward from the Out-Patient Department April 2. He spoke practically no English. The history was given by a friend who knew very little about it. What he gave was considered unreliable. The patient came because of loss of vision in the right eye.

For some weeks the patient had been less vigorous than usual. For some time he had had loss of vision in the right eye. Five days before admission he "caught cold" and had a series of possible chills. Three days before admission he vomited. Since that time he had not had a movement of the bowels, had seemed to have discomfort in the lower abdomen and some oliguria, and had eaten practically nothing.

No past history is recorded.

Clinical examination showed an obese, short-necked man with pendulous abdomen, relatively small limbs, and evidence of some loss of weight. He breathed rapidly but without apparent distress. Over the back were many pigmented keratoses. Most of the teeth were gone. There was some pyorrhea. The chest was barrel-shaped. The lung signs were as shown in the diagram. The location of the apex impulse of the heart is not recorded. The left border of dullness was 10 centimeters to the left of midsternum, a centimeter and a half outside the midclavicular line, right border 3.5 centimeters to the right, supracardiac dullness 7.5 centimeters. Sounds of fair quality at the base. Pulmonic second sound greater than aortic second and perhaps reduplicated. Rhythm regular. Rate rapid. No murmurs or thrill. Pulses and arteries normal. Blood pressure 115/70. Abdomen held somewhat rigid. Genitals and rectal examination showed nothing significant. Prostate symmetrically enlarged. Right pupil slightly irregular, no reaction, lens opaque. Left pupil contracted, reacted to light sluggishly. Right knee-jerk normal. Left knee-jerk and both ankle jerks not obtained.


Dullness to left of cardiac dullness. Bronchial breathing. No increased tactile fremitus or voice sounds.
Râles at both bases, back and front.
Area of slight dullness with diminished breath sounds of bronchovesicular quality. No increased tactile fremitus or voice sounds.

Urine: amount not recorded, specific gravity 1.020, a large trace of albumin, sediment loaded with hyalin and granular casts. Blood: 16,200 to 19,900 leukocytes, hemoglobin 75 per cent., reds 5,300,000. Hinton: insufficient blood for test. Non-protein nitrogen 90 milligrams.

Temperature 100° to 101°, rectal. Pulse 97 to 105. Respirations 26 to 32.

X-ray examination showed dullness at both bases. The outline of the diaphragm was not made out. On the left side the dullness rose as high as the midscapular region. The upper portion was mottled. The plates were unsatisfactory on account of motion and rotation.

The orders included rest in bed, a soft solid diet, digifolin and morphia.

The day after admission there were medium crepitant râles about the left axilla and back, fewer on the right. A very slight left knee-jerk was obtained, also ankle jerks and sluggish arm jerks. There was no Babinski or clonus.

April 4, after a day without apparent change in condition, the patient clutched desperately at his precordia and became cyanotic, dyspneic and unconscious, gasping and perspiring. The pulse could not be felt at the wrist. The heart sounds were inaudible. The blood pressure was not obtainable. An intracardial injection of ten minims of adrenalin was given, and also artificial respiration, both without effect. Within seven minutes after the onset of the seizure he died.

## DISCUSSION

BY RICHARD C. CABOT, M.D.

### NOTES ON THE HISTORY

If you can make a diagnosis from that history you are much sharper than I. I have no idea up to date what was the matter with that man.

### NOTES ON THE PHYSICAL EXAMINATION

The lung signs mean either bronchopneumonia or bronchial solidification from congestion.

They are more like pneumonia than anything else, but they are not the typical signs.

The râles are presumably the râles of edema.

DIFFERENTIAL DIAGNOSIS

We had better start at the end. We have very little evidence before that. We can guess at many classes of ills in the diagnosis of a case of sudden death apparently with pain, intense cyanosis and pulse failure. Two things especially should be thought of, (a) blocked coronary arteries with cardiac infarct and (b) pulmonary embolism with thrombosis. When we make the second of those diagnoses we need evidence of venous thrombosis elsewhere in the body whence an embolus might be washed off into the lungs; for instance varicose veins, or the thrombosis which takes place in the pelvis after a uterine operation. Cardiac infarct, especially at his age, is a little more reasonable if we go back over his history. He came into the hospital because he could not see. Over his right eye they found a cataract. That was considered of no importance. He had had it for a long time. The rest of his history is unimportant. The physical examination shows very little except a suspicion of pneumonia in the left lung and certainly passive congestion with fluid in both pleurae. I do not make anything out of his reflexes. We have not enough there to help us toward any diagnosis.

The best guess I can make is cardiac infarct, on the basis of arteriosclerosis with coronary sclerosis, failing heart, presumably from previous hypertension, and very possibly bronchopneumonia in the left lung.

A PHYSICIAN: How do you explain the albumin, casts and high non-protein nitrogen?

DR. CABOT: Albumin and casts when you have passive congestion do not mean anything more than passive congestion. We certainly have passive congestion here. But it is quite possible that there is a certain amount of arterial disease. I suspect there is some vascular nephritis, too.

INTERPRETATION OF X-RAY

The findings probably represent a pneumonic process in the lower left chest.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Bronchopneumonia.
Arteriosclerosis.
Coronary occlusion?

DR. RICHARD C. CABOT'S DIAGNOSIS

Arteriosclerosis, general.
Coronary sclerosis and occlusion.
Cardiac infarct.
Vascular nephritis?

ANATOMIC DIAGNOSES

1. *Primary disease.*

Coronary sclerosis and thrombosis.
Infarct of the heart.
Acute myocarditis.

2. *Secondary or terminal lesions.*

Central necrosis of the liver.
Arteriolar sclerosis, generalized.
Vascular nephritis, benign type.
Septicemia, streptococcus hemolyticus.

3. *Historical landmarks.*

Bilateral direct inguinal hernia.

DR. TRACY B. MALLORY: This man showed coronary occlusion and infarction. In fact he showed two infarcts. In the septal region was an old one that must have been of weeks' or months' standing. There was also a fresh thrombus in the descending branch of the left coronary. Many infarcts occur without pain, and we have no history suggesting the time of the onset of his older lesion. He had a hemolytic streptococcus septicemia at the time of death, and that probably had some connection with the terminal occlusion which he showed. It has even been claimed, though the evidence is to my mind inconclusive, that all coronary thrombosis is a result of infection. He had fairly general arteriosclerosis and a moderate degree of vascular nephritis, although I doubt that the kidneys played any great part in his symptoms.

The lungs were negative.

A PHYSICIAN: Was there any fluid in the chest?

DR. MALLORY: There was considerable fluid on the right.

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## CASE 15322

## DIAGNOSIS CONFUSED BY COINCIDENTAL INJURY

CHILDREN'S MEDICAL DEPARTMENT

A boy baby nine months old, of Norwegian parentage, entered May 15. The chief complaints were loss of weight and paralysis.

He was born at full term and normally delivered. The birth weight was nine pounds. He was breast fed for three weeks, then had milk, water and dextrimaltose. He took the mixture fairly well and seemed to be well nourished, weighing thirteen pounds at six months. He had no febrile illnesses.

When he was three months old his carriage blew over, throwing him out on his face. When he was picked up soon afterward his right knee was slightly red. A week later his right foot was somewhat swollen and purplish. A physician said this was due to scurvy brought on by scalding the milk. The swelling went down, but had not entirely cleared up at the time of admission. There is no mention in the history of treatment since the fall except that for three weeks before admission orange juice had been given. When the child was six months old it was found that touching or bending the right knee caused severe

pain. A month before admission limpness of the right leg was noticed. For twelve days he had been holding his head extended. For six days he had had fever. At admission the temperature was 105°. Spasticity of the arms was noticed four days before admission. Twice in the past four days he had seemed to suffer severe pain,—screwing up his face, rolling his eyes, stiffening out and shaking all over. At admission the arms were stiff.

He held up his head at six months. He never sat up because of pain on the attempt. During the past three weeks he had lost weight rapidly —one pound and two ounces in the past week.

His mother, father and two other children were living and well. The mother had had one miscarriage. There was no history of tuberculosis, diabetes, or hemophilia.

Clinical examination showed a poorly nourished child, quite pale, presenting a striking picture with his head greatly retracted, his back curved backward, his arms and left leg rigid and his fists tightly clenched. When turned on his back the body rested on the back of the head and the buttocks. The fontanel bulged. The pupils were alternately dilated and contracted, equal, did not react to light. The eyes deviated to the left. There was at times jerky nystagmus. The ears and throat were negative. The mucous membranes, lips and tongue were dry. The skin was very dry and inelastic. The neck was rigid. The lungs were clear. The heart was negative. The abdomen was flat. The spleen was not felt. Some small masses, however, were felt in the left lower quadrant, probably fecal. The arms and left leg were held rigid. The right leg was flaccid. The foot was rigid, but he could move the toes. The hands were clenched tightly, not in the carpopedal spasm of tetany. There was no Chvostek.

At admission the temperature was 105.4°, the pulse 100, the respirations 60. Later the temperature was 101.1° to 104.5°, the pulse 102 to 95.1, the respirations 40 to 71.

The child was at once given an enema and put upon chloral hydrate grains iv every four hours; ten per cent glucose solution, two ounces by rectum every two hours; and ten per cent karo solution, eight ounces every four hours. On the same day he was given 250 cubic centimeters of salt solution intraperitoneally and 85 cubic centimeters of ten per cent. glucose intrasinously. That day his blood count was 7,430,000 red cells, 18,800 leukocytes, 73 per cent polymorphonuclears, 25 per cent lymphocytes, 1 per cent basophiles, 1 per cent unclassified. The hemoglobin was 70. A lumbar puncture gave clear fluid under normal pressure, 62 cells, 50 per cent lymphocytes.

The day after admission he seemed better. He showed less stiffness and was able and inclined to cry a little. A right facial paralysis was now noticed. The temperature remained elevated, 103° to 104°. He was given 250 cubic centimeters of normal saline into the peritoneal cavity.

## Discussion

BY ELI C. ROMBERG, M.D

This case is interesting because it illustrates how a coincidental occurrence may prejudice the observer sufficiently to mislead him from the true situation. This child comes into the hospital in the middle of spring with the chief complaint of loss of weight and paralysis. Its past history is negative. We wonder why the child was breast fed only for three weeks and then put on the formula. We do not know whether the milk was of good quality or whether it was boiled. If the birth weight of nine pounds is correct, its weight of thirteen pounds at six months indicates that the child is definitely underweight.

The story of the baby's fall from its carriage and the swelling of the right knee soon afterwards is interesting. The physician who saw it soon afterwards thought it might be due to scurvy brought on by scalding the milk. We are not told whether the baby had any orange juice during this period. If the child had even as small an amount as fifteen to twenty drops a day, it would have been sufficient to prevent the occurrence of scurvy. Apparently however the swelling had gone on for months, because there still is some question of its presence at the time of admission, six months later. Scurvy appropriately treated ought to clear up in a week at least. If it persists in spite of treatment and the diagnosis has definitely been established, it may be due to the fact that extensive hemorrhage in the involved area has occurred. Absorption may have been slow and abscess formation has probably taken place, so that the problem is that of an abscess. Furthermore it is very unusual to have unilateral scurvy or involvement of only one "joint". It is true that in some cases superficial examination discloses the involvement of one limb or one "joint"; but if X-rays are taken one will discover some involvement on the other side. Here we note again that at six months the child suffered pain when his right knee was bent or touched and that he did not move that leg. This indicates some involvement of the extremities on that side either associated with the spine, hip, or knee, the pain either localized at one of these points or referred to them from a single focus. The history of temperature, generalized spasticity and convulsions, extension of the head and apparent pain suggests some meningeal involvement. Apparently the child has never sat up because of this pain, and there is a history of rapid loss of weight.

The physical examination of the child presents a picture essentially that of cerebrospinal system involvement, with opisthotonos, spas-

ticity, bulging fontanel and involvement of the eyes. Otherwise there was nothing interesting in the physical examination. Because the child was apparently dehydrated, he was given fluids in various forms. He was not able to hold very much by rectum, so that other means of fluid feedings were utilized. Normal saline was introduced intraperitoneally and glucose intrasinously in the hope probably in that way to encourage the rapid absorption of salt into the circulation. It is not a good policy to use the sinus for the introduction of fluids, particularly in a case like this where a child apparently has enough trouble under his cranium. It is difficult to know whether the needle is still in the sinus unless one frequently withdraws the piston to obtain a flow of blood and assure himself that it is still there. In such children, where dehydration has occurred, the sinus frequently is collapsed and it is difficult even to obtain blood from it, still more to introduce fluid into it. It would I think have been better to try the jugular, scalp, or some other superficial vessel. The introduction of irritating drugs or blood should absolutely be avoided because of the difficulties I have just named. I do not think it is a very good policy to use the fontanel for the obtaining of blood for Wassermanns unless it is absolutely impossible to get it elsewhere. Most of the scalps of children are unclean, and unless the scalp is shaved and thoroughly cleansed one may possibly introduce some infection intracranially. The child's red count is high probably because of circulatory stasis in this constantly rigid region, or because a blood droplet was forced out, bringing about a concentrated mixture. The white count is slightly elevated and there is a high polymorphonuclear count. The lumbar puncture shows definite increase of cells and apparently clear fluid. Then the child temporarily becomes better, because his spasticity is relaxed and he seems a little more animated. The temperature, however, persists. Another lumbar puncture confirms the previous findings. Probably the child was given calcium chloride because someone must have considered a latent tetany, although in the presence of such positive spinal findings I do not see why it was given.

Summarizing the situation, then, we have a child who at three months of age develops a sore limb which persists for six months. For about two weeks before death he has generalized convulsions and involvement of his head and all extremities, associated with a temperature and a positive spinal fluid. What are the conditions that might cause such a picture? Suppose the condition could be associated with the fall and the child received some bruise through which an organism might enter. One ought to consider tetanus; but the fact that his nervous system was not involved until six months later rules out this condition, because the incubation of tetanus is from six to twenty days. What impresses me particularly is that the child has been irritable and fussy for the past six months, that he has been underweight and recently has lost weight rapidly. It then suggests an insidious chronic disease with a fulminating terminal picture rather than an acute process. In childhood tuberculosis is the main offender. The portal of entry may be from the mouth and throat and the infection then find its way to the various glands. If any caseation takes place it is taken up either through the circulation or by the lymphatics, then finding its resting place in tissues attractive to the organism, like lymphoid tissue or cancellous areas of the bones. These conditions may be quiescent for a long time and may go on without presenting any difficulty at all, or the disease may develop later in life, or it may be precipitated in an acute form by disease or injury. There is still some contention whether injury plays an important part in spreading a tuberculous lesion. If we make a diagnosis of tuberculosis in this case we probably shall have to consider the involvement of the leg as of tuberculous origin, particular because of its chronicity. Something apparently occurred which activated some focus of tuberculosis in this child, throwing the organisms into the general circulation and depositing foci throughout the body, so that the temperature, convulsions, and apparent meningeal involvement are but secondary and terminal manifestations of the disease. The fact that nothing was found in examination of the lungs does not exclude miliary tuberculosis, because very frequently the condition is recognized by X-ray or by autopsy. The leukocyte count is rather high for tuberculosis, but it may be affected by some associated infection which would bring up the count. Ordinarily tuberculosis gives a reduction in leukocytes, but in tuberculous meningitis the count may be definitely increased and there may be actual leukocytosis as high as 25,000 in the terminal stage of the disease.

### FURTHER HISTORY

Another lumbar puncture gave 20 cubic centimeters of clear fluid under increased pressure, 68 cells, about 50 per cent polymorphonuclears and 50 per cent lymphocytes; some of the cells could not be positively identified. The fluid showed no sugar, protein much increased, globulin positive. A smear showed tubercle bacilli.

The child was given grains x of calcium chloride every four hours. On May 17 the temperature was 103°. He died early that morning, having been in the hospital a little over two days.

### FURTHER DISCUSSION

The spinal fluid in tuberculous meningitis generally is under increased pressure. When it is withdrawn it appears clear, but when com-

pared with water is slightly cloudy. There is a predominance of mononuclear cells. If the fluid is allowed to stand for several hours a pellicle is generally formed in which the tubercle bacilli may be found. During the early stages of the disease it may be difficult to discover the bacilli unless one is very persevering, but generally at the end of the disease they may be found in great numbers. The finding of these organisms in the spinal fluid definitely establishes the diagnosis of tuberculous meningitis.

Naturally there is nothing that we can do in such a condition except make the child a little more comfortable. What are the main difficulties to contend with? First convulsions, then vomiting, and unwillingness or inability to take food or fluids. For the convulsions one can resort to frequent spinal punctures to relieve pressure. Small doses of sedatives of some sort can be used.

If the child refuses to take fluids by mouth or cannot because of vomiting, rectal feedings or subcutaneous and intraperitoneal injections of salt can be given. Occasionally if the disease lasts a long time and one has used the peritoneum and skin much more than one would like, if absorption seems to be slow and the skin is in a bad condition, one can resort to tube feedings, giving to the child milk, soups, or eggnogs.

The outcome is hopeless. At times the child may seem to improve, brighten up, sit up and even talk, and one may feel that one has made a wrong diagnosis. But this improvement is a part of the picture of this disease. The improvement is but temporary. The child lapses into his old state and progressively becomes worse. If the case is a fulminating one the whole process may terminate in a few days. The slow, insidious, prolonged prodromal state may last several months; but after the acute process begins it is but a matter of weeks before death occurs.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Tuberculous meningitis.
Malnutrition.

ANATOMIC DIAGNOSES

Tuberculous ulcers of the intestine and appendix.
Tuberculosis of the mesenteric and retroperitoneal glands.
Tuberculous meningitis.
Solitary tubercle of the brain.
Miliary tuberculosis of the lungs, liver and spleen.
Hemorrhagic necrosis of the wall of the esophagus with perforation.
Fibrinous pleuritis, right.
Otitis media, left.

DR. TRACY B. MALLORY: The autopsy showed along the base of the brain and in the fissures of Sylvius a fairly thick layer of exudate beneath the arachnoid. In this could be recognized in gross numerous minute points suggesting tubercles. In the region of the optic thalamus on the right a somewhat larger, caseous "solitary tubercle" was found. Miliary tubercles were present in the majority of the other organs.

It is always interesting in these cases to attempt to trace the origin of the infection. In this case no old lesions were found in the lungs or the bronchial or cervical lymph nodes. Tuberculous ulcers were however present in the small intestine, and the mesenteric lymph nodes were greatly enlarged and caseous. There seems no doubt therefore that the organisms entered by the intestinal tract. No effort was made to determine whether the organisms were of the human or the bovine type. The great majority of the cases of intestinal tuberculosis are, however, bovine, and this baby was fed on cow's milk after the first three weeks.

DR. ROMBERG: It is surprising to see the extensive involvement of tuberculosis in this child. The absence of intestinal symptoms is very unusual, particularly when one considers that the portal of entry was the intestinal tract. These areas were probably involved for a long time, as indicated by the degree of ulceration and necrosis. Frequently one finds in the brain minute tubercles which are fairly well walled in and cause no apparent difficulty. At other times a tubercle may be so large that it presents all the intracranial symptoms of a disturbing tumor mass.

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## A TEMPEST IN A TEAPOT

IT must be obvious to those who are at all acquainted with the facts that Secretary of the Interior Wilbur made a decided faux pas last week in comparing child welfare work in the North and East unfavorably with that in the South. Dots on a map give a useful synoptic view of a situation if one knows what the dots mean; otherwise they may create confusion in the minds of the listeners. County health organization may be more perfect in the South than it is in Massachusetts, because in the South the county is the important political unit and in the more settled North it is relatively unimportant. Government bureaus may be more active in the South than in the North because in the South they are needed to carry on the work which in the North is carried on by State, town, and a multiplicity of private agencies.

If dots on a map represent only county organizations or federal activities, they will naturally be more numerous in the southern section than in the northern, but if they represent actual accomplishment in the given field the North will receive its full share, for the proof of the pudding is in the eating.

It may be natural, but unwise, for high government officials to think too much in terms of sterotyped formulae, for formulae may not always meet most adequately varying conditions of need. This, of course, is the greatest drawback to bureaucracy. It does not cut its coat to fit the cloth, or even the individual. It cuts its coat and tries to make the individual conform to it. We in New England do not feel satisfied with our child welfare organization. Far from it. We realize that much more may be done, but we know that much has been done and that we are progressing and that our work compares favorably with that in other States. We do not feel that the millennium will be reached any sooner by organization along county lines and we regret that Secretary Wilbur has seemed to put us in a bad light and himself in an awkward position by what was unquestionably an ill-considered and casual remark.

## USEFULNESS OF A "PRECEDENT BOOK"

IN many modern hospitals it has been found useful to keep a record, a so-called "Precedent Book", of established practice in the use of new methods of treatment which makes available the details of such treatment for use by the members of the staff, their assistants, and house officers. Such a precedent book has become almost an essential in recent years owing to the rapidity with which advances are made and new methods become available in medical treatment. It has several advantages.

The technical details of newer procedures are often difficult to carry in mind, and yet accuracy and exactness are essential to success in using them. The "Precedent Book" gives such details in the form accepted by the staff through a committee appointed to pass upon and recommend such procedures for adoption. The special experience of some members of the staff may be called upon in formulating the recommended treatment for certain conditions and so by a coöperative arrangement the hospital as a whole profits. The attempt to put into written form a description of a therapeutic procedure encourages simplification, criticism, and efforts to improve methods altogether in keeping with the principles of efficiency and economy expressed by President Hoover. Furthermore, additional security is afforded both to the physician individually responsible for the physician, and to the hospital, when it is possible to carry out treatment by methods which have been so thoroughly tested, although new, as to be accepted by the staff as a whole.

Today the dietary treatment of certain common diseases lends itself most strikingly to such a treatment. Rapid progress has been made, and yet few physicians have the time to devote to a special and intensive study of this subject. In diabetes, the dietary treatment has been simplified by the adoption in certain diabetic

clinics of a few standard diets. Emergencies arise in the course of diabetic treatment, notably acidosis, or surgical conditions, which require very specialized management. An outline for the treatment of such conditions might well be included in the "Precedent Book." Precautions to be observed in the giving of subpectoral infusions or of intravenous treatment, an outline of treatment for duodenal ulcer, the treatment for such an emergency as mercury bichloride poisoning, the procedure to be followed by the house officer in various emergencies, and many other subjects would be suitable for consideration by the hospital staff in such a "Precedent Book". Once begun the "Precedent Book" will be continued because its usefulness will be appreciated.

## ARE BARBER SHOPS A MENACE TO HEALTH?

For several years the legislature has been asked to make laws regulating the practice of barbering. The contention has been made that many of the shops are unsanitary and that diseases have been contracted in these establishments.

The State Department of Public Health has been directed to ascertain the facts and has issued a questionnaire and conducted one hearing.

It is reported that the Department has found little evidence that diseases have been contracted in barber shops. Even with this lack of evidence of danger it would be comforting to know that ordinary cleanliness is required of those who are engaged in this service. Perhaps the end can be secured if the public will discriminate and only seek well managed barber shops.

## THIS WEEK'S ISSUE

Contains articles by the following named authors:

Dayton, Neil A. M.D. Ohio State University, 1915. Director of Research, Massachusetts State Dept. of Mental Diseases. Member, American Psychiatric Association; Massachusetts Psychiatric Association; New England Society of Psychiatry; American Association Study of Feeble-minded; American Statistical Association. His subject is: "The Relationship Between Physical Defects and Intelligence." Page 245. Address: 109 State House, Boston.

Ohler, W. Richard. S.B., M.D. Harvard 1914. Instructor in Medicine, Harvard. Assistant Visiting Physician, Boston City Hospital. His subject is: "The Care of the Surgical Diabetic." Page 259. Address: 270 Commonwealth Ave., Boston.

Hill, Lewis Webb. A.B., M.D. Harvard 1913. Assistant Visiting Physician to Children's Hospital. Instructor in Pediatrics, Harvard Medical School. Visiting Physician, Boston Nursery for Blind Babies. His subject is: "Nutritional Anemia in Infancy." Page 261. Address: 483 Beacon St., Boston.

Sowles, Horace K. A.B., M.D. Harvard 1915. F.A.C.S. Out-patient Surgeon, Massachusetts General Hospital. Assistant Surgeon, Faulkner Hospital and Beth Israel Hospital. Consulting Surgeon, Lawrence Memorial Hospital. Instructor in Surgery, Tufts Medical School and Harvard Medical School. His subject is: "Acute Yellow Atrophy; the Incidence of the Disease and the Report of a Case." Page 265. Address: 279 Clarendon St., Boston.

Rosenberg, Joel. M.D. Tufts Medical School, 1926. Resident Surgeon, Beth Israel Hospital. His subject is: "Medullary Carcinoma of the Ovary in a Girl Twelve Years Old." Page 268. Address: 470 Warren St., Roxbury, Mass.

Cameron, Walter C. B.S., M.D. University of Pennsylvania, 1927. Eye House Officer at Massachusetts Eye and Ear Infirmary. His subject is: "Notes on Some Eye Accidents of July 4th." Page 272. Address: Massachusetts Eye and Ear Infirmary, Boston.

Balch, Franklin G. A.B., A.M., M.D. Harvard, 1892. F.A.C.S. President of the Boston Medical Milk Commission. Surgeon, Massachusetts General Hospital; Lawrence Memorial Hospital, Medford, and Faulkner Hospital, Jamaica Plain. His subject is: "Certified Milk in Honolulu." Page 273. Address: 279 Clarendon St., Boston.

Hodgkins, E. M. M.D., Tufts College Medical School, 1915. F.A.C.S. Assistant Professor of Surgery, Tufts College Medical School. Surgeon-in-Chief, Roxbury Hospital. His subject is: "A Double Adjustable Knife for Cutting Fascial Strips." Page 274. Address: 12 Bay State Road, Boston.

Seybolt, Robert Francis. A.M., Ph.B., Ph.D. Professor of the History of Education, University of Illinois, since 1926; Harvard University 1925-26 (first semester) and summer sessions of 1926, 1927 and 1929. His subject is: "A Cure for Cancer in 1720." Page 275. Address: University of Illinois, Urbana, Illinois.

## MISCELLANY

### MORBIDITY IN THE RECENT INFLUENZA EPIDEMIC

**The United States Public Health Service has recently announced the results of surveys in certain cities concerning the influenza epidemic of 1928-29. Although full analysis of the data collected will require considerable time, some preliminary tabulations have been made which suffice to show approximately the gross morbidity rates in the several localities studied.**

Because of the almost prohibitive difficulties and expense of canvassing rural communities, the surveys were limited to large cities; and these were chosen with a view to representing as many sections of the United States as practicable. Those selected were as follows: San Francisco and Seattle, on the Pacific Coast; Des Moines, Kansas City (Mo.), Cincinnati, and Pittsburgh, representing the Middle West; New Orleans, as a southern city, and in the northeast, Baltimore, Syracuse, and Boston. It was later found practicable to add a single small city, Farmington, Mo.

In order to afford sufficient numbers for a statistical analysis, the sample canvassed in each locality (except Farmington) comprised not less than 10,000 persons, and in cities of more than 400,000 inhabitants, this number was increased to 15,000 or more. To secure a representative sample a total of 12 to 20 districts was marked off in each city, and in each of these districts a systematic house-to-house canvass was made.

It is fully recognized, however, that all records are subject to certain obvious but practically inevitable errors, due principally to lack of definite criteria for the diagnosis of influenza, and to lapses of memory on the part of the householders from whom the information was obtained. These causes tend, respectively, to uncertainty in the diagnostic classification of reported cases, and to omission of a certain proportion of cases, especially those which were not of sufficient severity to leave a lasting impression upon the memory.

As regards diagnostic classification of cases, enumerators were instructed to inquire concerning the occurrence of "influenza," "grippe," "pneumonia," or "colds," and to record for each case the diagnosis given by the householder. Presumably all the cases reported as pneumonia had been so diagnosed by attending physicians; and a preliminary check of the records indicates that this was true also in a large proportion of the cases classed as "influenza" or "grippe." Also it is probable that the cases so diagnosed were usually of sufficient severity to be remembered by the householders and reported to the enumerators. Therefore, with full recognition of all the probable sources of error in reporting, it is believed that the records as to these classes of cases have a fairly definite qualitative and quantitative significance.

On the other hand, a large majority of the cases reported as "colds" had not been attended by physicians; and it is well known that ordinary colds are likely to be forgotten within a few weeks. Hence, it may be inferred that the record of colds is far from complete, and that it probably includes cases of a more or less heterogeneous type.

The total number of cases of influenza and grippe, pneumonia, and colds per 100 persons canvassed for the several States, was as follows: San Francisco 34.1; Seattle 30.9; Kansas City (Mo.) 27.1; Farmington, Mo. 37.7; Des Moines 47.1; New Orleans 29.6; Cincinnati 27.0; Pittsburgh 25.2; Baltimore 15.1 (exclusive of colds not confining the patient to bed); Syracuse 26.8; Boston 23.5.

The average of all these localities except Baltimore was 29.7 cases of influenza, grippe, pneumonia and colds per 100 persons canvassed. Nearly 15 per cent. of the population canvassed in all localities surveyed in 1929 gave a history of having suffered attacks of influenza or grippe, while 0.47 per cent. gave a history of pneumonia and an additional 14 per cent. reported "colds" which may or may not have been directly related to the epidemic.

The incidence of pneumonia is shown to be about one-third as high in the epidemic of 1928-29 as in that of 1918. It is probable that the disparity between the two epidemics is greater than these figures would seem to indicate since the periods covered by the 1928-29 record are generally longer than those covered in 1918. Moreover the 1918 epidemic occurred at a season when the prevalence of pneumonia is normally rather low, whereas the recent epidemic occurred at a season when the prevalence of pneumonia is normally rather high and increasing. Mortality records indicate a much greater disparity between the 1918 and 1928-29 epidemics; apparently the cases in the latter epidemic were of a much milder type.

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## PHYSICIANS URGED TO BE CAREFUL

### When Executing Medical Certificates on Civil Service Applications

*(Statement of the United States Civil Service Commission)*

The Government, and applicants for examinations themselves, are frequently embarrassed by the improper execution of medical certificates attached to applications for Federal civil service examinations.

New appointees are required to undergo a physical examination by a Government medical officer before entering upon duty. For many examinations a preliminary medical certificate is required in connection with the application for examination, for consideration in determining eligibility for examination and in some cases for rating on the element of physical ability. Frequently, Government medical officers find in the examination at the time of appointment physical disqualifications which must have existed when the preliminary medical certificate was executed by the private practitioner, although no mention of such physical defects is found in the private practitioner's medical certificate. Such a situation presents a problem to the Government, especially if the appointee has traveled a considerable distance to accept the appointment. In many cases the appointment must be canceled, with resulting loss of time and money to the disappointed applicant.

The Civil Service Commission feels that these discrepancies between medical certificates executed by private practitioners and those made later by Gorernment medical officers are due in some cases to carelessness upon the part of the private practitioners and in others to a liberal attitude deliberately assumed in the mistaken belief that by ignoring or minimizing physical defects the applicant is assisted in obtaining employment.

The Civil Service Commission's forms for medical certificates attached to application blanks are comprehensive and clear. If all private practitioners will exercise due care when filling out the certificates they will not only render a service to the Government but will also give the maximum service to the applicant who pays the fee for the preliminary physical examination.

## THE WORLD'S CHILDREN

### MURRY AND LEONIE GUGGENHEIM FOUNDATION

The first project of the newly created Murry and Leonie Guggenheim Foundation is the immediate building of a free dental clinic for children in New York City. The cost of this is estimated at $3,000,000 to $4,000,000. In addition to preventive and reparative work there will be provision for the training of dental hygienists for service in public schools and industrial establishments. The immediate aim of the foundation is to provide such clinics for New York, but its avowed ultimate purpose is "the promotion through charitable and benevolent activities of the well-being of mankind throughout the world."

### MENTAL HYGIENE CLINICS

Connecticut has added a full-time psychiatrist to its bureau of mental hygiene in the State department of health, which will expand its program to include child-guidance work.

The New Hampshire State Board of Health held its first behavior clinic at Durham, June 5 in connection with the regular child-health conference for babies and children of preschool age. The parents showed very great interest in the behavior clinic, and the board of health is planning to hold similar clinics in all parts of the State in connection with the child-hygiene work.—*Children's Bureau.*

## THE HEALTH OF IMMIGRANTS

Formerly immigrants to the United States had to appear at the port of entry before knowing whether their health conditions were satisfactory and that they would be permitted to land. This involved hardship, expense and delay. Those denied admittance to the country were sometimes pauperized and broken in spirit.

Under present conditions immigrants are more generally examined at the port of departure, thereby weeding out those with diseases which would be a menace to our people and giving those with temporary and curable diseases time to recover before embarkation.

The advantages to this country and the applicant are obvious. Prior to 1882 many mentally and physically diseased persons got into the country, some requiring hospitalization; the more troublesome cases finally getting into institutions for the insane. Even as late as 1890 several recognized insane and idiotic persons were allowed to land.

Dr. Taliaferro Clark, Senior Surgeon, United States Public Health Service reports in the *United States Daily* for July 26, 1929 interesting statutes bearing on this subject.

## REMEDY FOR CANCER ELUSIVE

On April 9, 1928 Surgeon General Cumming arranged a conference of consultants to advise him regarding the Cancer program of the Public Health Service. Since that time the commerce subcommittee under the chairmanship of Senator Jones has written to all who attended the Conference asking for opinions with respect to the report of the Surgeon General especially soliciting further suggestions.

Communications received from Johns Hopkins, Columbia, Cornell and Yale tend to emphasize the difficulties in the way of finding a cure for cancer with the prediction that any hope for an early solution of the problem is premature.

## THE EVENTUAL ELIMINATION OF LEPROSY FROM THE UNITED STATES

Surgeon General Cumming is reported as hopeful that leprosy may be eradicated in this country through more complete knowledge of the beneficial properties of chaulmoogra oil.

Dr. N. E. Wayson of the Leprosy Investigation Station in Hawaii has been using iodine combined with chaulmoogra oil.

Dr. Cumming reports that three more patients are to be released from the leprosarium at Carville, La., and further says that "the disease is gradually being conquered."

## MAINE MORTALITY STATISTICS: 1928

Washington, D. C., June 25, 1929.—The Department of Commerce announces that the 1928 death rate for Maine was 1,384.3 per 100,000 population as compared with 1,381.8 in 1927.

The principal increases in rates (per 100,000 population) from those of the preceding year were from: nephritis (103.3 to 110.3), diseases of the heart (260.5 to 264.8), cerebral hemorrhage and softening (140.4 to 142.6), and diabetes mellitus (21.3 to 24.8). The death rate from all accidental causes increased from 68.9 to 80.3, the individual type of accident showing the greatest increase being accidental drowning (8.6 to 14.3).

Significant among the decreases in rates from 1927 to 1928 were those from: cancer (137.2 to 134.5), tuberculosis, all forms (68.3 to 60.3), diarrhea and enteritis, under 2 years (18.7 to 15.0), scarlet fever (4.5 to 2.1), whooping cough (9.5 to 6.8), measles (10.1 to 4.8), and influenza (38.6 to 35.0).

The estimated population in 1928 was 795,000 and in 1927 was 793,000.—*Bureau of the Census.*

## CLAIM FOR A NEW ANAESTHETIC

It is reported that the faculty of the dental school of Northwestern University have perfected an anaesthetic which will produce its effect in much less time than that of other drugs hitherto in use.

This anaesthetic is ethyocaine borate and has been used on nearly a thousand patients in the dental school clinic. The advantages seem to be in addition to rapidity of action, less toxicity, absence of post-operative pain and general systematic disturbances.

## PHYSICAL MEASUREMENTS OF 30,000 BOYS AND GIRLS

The results of physical measurements made by the United States Public Health Service of nearly 30,000 school children of native white parents and grandparents in the United States have recently been announced. The measurements made were standing height or stature, sitting height, or trunk length,

weight, chest circumference (at rest), transverse chest diameter, or width of the chest, antero-posterior diameter, or depth of the chest, and vital capacity. The method of making the measurements was identical in all places, the measurements all being made by three medical officers of the Public Health Service. The children were, roughly, from four geographic sections of the United States—Northeast, North Central, South Central, and Western States.

As age increases, weight and vital capacity increase more rapidly than any of the other measurements taken.

In all the measurements except vital capacity and transverse chest diameter there is a period of from 2 to 5 years between 11 and 15 years of age, inclusive, during which the mean measurement for girls exceeds the mean for boys of a corresponding age. In general, the largest excesses of girls over boys occur at about 13 years of age.

By computing the difference between the mean measurements at successive ages we may approximate the mean annual increments of growth in these measurements. The age of greatest growth varies somewhat for the different measurements, but there is a tendency for a maximum percentage increment about the age of puberty, the maxima for girls coming 1 or 2 years earlier than the maxima for boys.

For every measurement considered in this study there is a period of three or more years between 6 and 13 years of age during which the mean annual increment for girls exceeds the increment for boys of a corresponding age. In general, the largest excesses in the increments of girls over those of boys occur from 11 to 12 years of age. The increment from 13 to 14 years of age is for every measurement larger for boys than for girls.

The mean annual increments of girls exceed those of boys at considerably earlier ages than in the case of the mean measurements themselves.—*United States Public Health Service.*

## CORRESPONDENCE

### GOLDSMITH'S COMMENTS ON AVENBRUGGER'S BOOK ON PERCUSSION

August 2nd, 1929.

To the Editor of the NEW ENGLAND JOURNAL OF MEDICINE:

When Avenbrugger first published his book on "Percussion", Oliver Goldsmith was doing hack work in London for John Newbery, a bookseller in St. Paul's Churchyard. Newbery had started, in 1761, a newspaper, the "Public Ledger", and Goldsmith contributed papers twice a week to it. Although Goldsmith was definitely settled on his career as an author-poet, he had not forgotten his medical education and his long trip abroad which ended in a degree of M.B. from either Louvain or Padua in 1756. It is, therefore, not surprising that he was at once interested and probably appreciated the importance of Avenbrugger's discovery. He wrote the following note to the editor of the "Public Ledger", which was published August 27, 1761. This letter of Goldsmith's was discovered in the files of the "Public Ledger" by Professor Ronald S. Crane and it appears in his "New Essays by Oliver Goldsmith", Chicago, University of Chicago Press, 1927. I am indebted to Dr. John F. Fulton for calling to my attention this interesting letter of Goldsmith's which appears below.

Very truly yours,
HENRY R. VIETS, M.D.

AVENBRUGGER'S DISCOVERY OF PERCUSSION[1]

*To the Compiler of the PUBLIC LEDGER*

SIR,

They who trust to foreign literary journals for a character of foreign publications will probably be deceived; in them we find every book well written, and every author ingenious; we must consult the works themselves if we would form a judgment.

As I flatter myself that I shall have many of those publications, almost as soon as the journalists in question, any judgment I am capable of forming, will at least be unbiassed by former authority.

There has been just published at Vienna,[2] a Latin treatise with the following title, "Leopoldi regionnosocomio, nationum Hispanica Medici Ordinarii, inventum novum expercussione thoracis humani ut signo abstrusos interni pectoris morbos detegendi: Or, a new invention for the discovery of latent disorders in the breast, by striking the thorax; by Leopold Avenbrugger, M.D. & c."[3].

I have not, says our medical adventurer, been incited to this publication by an itch for writing, nor the delusive pleasure of speculation, but an experience of seven years has confirmed my opinion, and improved my practice in this discovery. He continues to observe, that the thorax (or that part of the body which lies under the upper ribs and breast bone) when struck by the tops of the fingers, armed with a glove, sounds somewhat like a drum when covered with a woollen cloth; this sound is in every part pretty nearly equal except just over the heart. In lean men the sound is more perceptible than in fat, and the latter require a stronger blow to excite it. This sound he affirms to be one principal criterion of the state of the thorax, and in those who have a latent disorder there, the difference of sound is easily perceptible.

To examine the patient properly, it is necessary previously to cover the breast with a linen cloth, or to use a glove on the hand which examines; and while the Physician gently taps with his fingers, the patient is to be desired to fetch an inspiration, then to breathe, and so forth, till the variation is perfectly perceptible. If the breast be struck, he is to hold his head erect, his shoulders thrown backward; if the side is to be examined, he is to lift both his arms to his head; if his back, then his head is to be bowed forward, and his shoulders drawn down to his breast, by these means the sound will be more distinct and perceptible.

To know the difference between the sounds which the thorax returns when in an healthy state, and when afflicted with any latent internal disorder; let a man first strike his own thorax, and then strike his thigh; and he will perceive that one has an hollow, and the other a kind of dumb fleshy sound; the same difference will appear between an healthy and a disordered breast; the healthy thorax will

[1]From the *Public Ledger*, August 27, 1761.
[2]The title-page is dated 1761.
[3]There are two misprints in the title as given here: for "Hispanica" read "Hispanico," and for "expercussione" read "ex percussione." The analysis that follows, though very brief, is in general sufficiently accurate.

return the hollow sound described above, the infirm thorax the dumb fleshy sound, generally resembling that of the stricken thigh. To be perfect in this method of distinguishing, the practitioner should examine several breasts, and he will discover the minutest deviations. From this method therefore alone, says my author, many disorders may be detected, which it were impossible by any other pathognomonic symptom to discover. For if any particular part of the thorax gives either a more obscure or a louder sound than might be expected, such a difference is probably the result of latent disorder, and the infirmity lies under the part returning that difference of sound. From these premisses confirmed by experience, he has deduced the following observations, 1st, The more fleshy the sound, the more dangerous the disease. 2dly, The greater space of the breast the fleshy sound occupies, the greater the patient's danger. 3dly, If the patient's left side be perceived to thus affected, it is more dangerous than an affection of the right. 4thly, The upper part of the thorax thus affected, is less dangerous than the lower part. 5thly, The fore part is less dangerous than the hinder part. 6thly, If the whole thorax be deprived of its sound, the symptom is certainly fatal. 7thly, If the breast-bone return no sound, it is fatal. 8thly, If the part which covers the heart returns the fleshy sound, it denotes the death of the patient.

Such are the outlines of this new discovery: whether it may be of use to society or not, there is no necessity for me to pretend to determine, only this may be observed, that the lungs are often even in the most healthy state, found to adhere to the pleura, and in such a case, I fancy the sound would, in that part, deceive the practitioner; however, I shall not pretend to set my conjecture against his experience. Upon the whole, it is a trial that may be easily made, and to borrow an expression from Doctor Rock,[4] *If it cannot cure, it can do you no harm.*

I am, Sir, & c.

[4]A well-known quack, whom Goldsmith had twice ridiculed in the *Citizen of the World.* See Letters LXVIII (August 25, 1760) and CXIII (April 14, 1761).

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## ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

535 North Dearborn Street, Chicago, Ill.,
July 29, 1929.

Managing Editor,
The New England Journal of Medicine:

In addition to the articles enumerated in our letter of June 29 the following have been accepted:

Abbott Laboratories
Viosterol—Abbott

Benzol Products Co.
Neocinchophen—B. P. C.

Dick X-Ray Co.
I-X Barium Meal

Parke, Davis & Co.
Parke, Davis & Co.'s Viosterol

E. R. Squibb & Sons
Viosterol Squibb 100 D
Squibb's Viosterol Cod-Liver Oil 5 D
Squibb's Viosterol Cod-Liver Oil 5 D Mint-Flavored

Terrell's Laboratories
Rabies Vaccine Phenolized, Terrell

Yours truly,
W. A. Puckner, *Secretary,*
Council on Pharmacy and Chemistry.

## NEWS ITEMS

APPOINTMENTS AT TUFTS COLLEGE MEDICAL SCHOOL—Dr. Harry J. Inglis of 43 Bay State Road has been appointed Professor of Oto-Laryngology in the Tufts College Medical School.

Dr. Inglis is a graduate of Wesleyan University and Harvard Medical School, graduating from the latter with the Class of 1904.

He is Surgeon-in-Chief of the Ear, Nose and Throat Department, Boston Dispensary, and a member of the following societies: American Medical Association; American College of Surgeons; American Laryngological, Rhinological and Otological Society; American Academy of Ophthalmology and Oto-Laryngology; New England Otological and Laryngological Society; Massachusetts Medical Society.

Dr. William E. Chenery, of 377 Commonwealth Ave., former Professor of Laryngology, has been made Professor Emeritus.

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DR. THEOBALD SMITH'S BIRTHDAY—On July 31 ult. Dr. Theobald Smith reached the age of seventy. This brilliant but modest man has devoted his life to the service of humanity. He honored Boston by working for several years as Professor of Comparative Pathology at the Harvard Medical School and Director of the Pathological Laboratory of the Massachusetts State Board of Health.

Since 1915 he has been Director of Animal Pathology at The Rockefeller Institute for Medical Research.

The quality of his mind and the devotion to the problems before him have led to the characterization of The True Scientist.

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TYPHOID FEVER IN LYNN—A number of cases of typhoid fever have been reported in Lynn, Mass., and the health officers are trying to find the source of the infection.

## NOTICES

### ASSIGNMENT OF COMMISSIONED AND OTHER OFFICERS OF THE UNITED STATES PUBLIC HEALTH SERVICE TO BOSTON

Assistant Surgeon (R) J. W. Bryan. Relieved from duty at Marine Hospital, New Orleans, La. and assigned to duty at Marine Hospital, Boston, Mass.

Professor Carl Voegtlin, Pharmacologist S. M. Rosenthal and Pharmacologist Floyd DeEds. Directed to proceed from Washington, D. C. to Boston, Mass. and return, for the purpose of attending the thirteenth International Physiological Congress, August 19-23.—*U. S. Public Health Service.*

## UNITED STATES CIVIL SERVICE EXAMINATION

### Physiotherapy Aide

Applications must be on file with the U. S. Civil Service Commission at Washington, D. C., not later than September 10, 1929.

Persons who enter this examination will not be admitted to any other examination for which the receipt of applications closes on the same date. The date for assembling of competitors will be stated on their admission cards, and will be about fifteen days after the close of receipt of applications.

The United States Civil Service Commission announces open competitive examinations under the above title to be held at any of the places listed in the circular at which examination is requested in applications received by the Commission at Washington, D. C., on or before the date mentioned above. Vacancies in the Field Service of the United States Veterans' Bureau and United States Public Health Service, and in positions requiring similar qualifications, will be filled from this examination, unless it is found in the interest of the service to fill any vacancy by reinstatement, transfer, or promotion.

Applications—Form 2600, which is required, may be secured from the following (the title of the examination desired should be stated): The U. S. Civil Service Commission, Washington, D. C., or the Secretary of the U. S. Civil Service Board at any examination place listed.

Form 2600 should be properly executed, including the medical certificate, but excluding the officer's certificate of residence, and must be on file with the U. S. Civil Service Commission at Washington, D. C., not later than the date indicated above.

The exact title of the examination as given at the head of this announcement, must be stated in the application form.

Preference—Applicants claiming preference because of military or naval service should attach to their applications the papers relative to such service called for in the application form.

## REPORT AND NOTICE OF MEETINGS

### THE MASSACHUSETTS ASSOCIATION OF BOARDS OF HEALTH

The regularly quarterly meeting of the Massachusetts Association of Boards of Health was called to order at noon on July 25 at the Walker-Gordon Farm, Charles River, Mass., the company being the guests of the Farm.

The subject of the afternoon was naturally related to milk, and the papers were supplemented by inspection of the plant, the cows, the process of milking and the subsequent treatment of the milk in preparing it for the customers.

The first paper was presented by Mr. Frank E. Mott, Milk Inspector of Boston, and was a review of existing conditions, discussing pasteurization and the production of raw milk under the care that permits it to be termed "certified."

Mr. Henry W. Jeffers, President of the Walker-Gordon Laboratory Co., spoke on the "Methods of Control and Production of Certified Milk." His story was extemporaneous, dealing with sanitary and business features, and in response to a call from the floor, he discussed later some items in the development of the technique, including the "merry-go-round."

Mr. Jeffers had high praise for the Boston Health Department, which since 1891 has given its hearty co-öperation towards the making of a pure, high-grade product. Such assistance makes it possible to go even further in matters of technique and economy towards the highest standards, and from an experience of some thirty years, the speaker believes that these standards are practicable. From a beginning with forty cows this plant at Charles River has now some 400, and the size of the plant has approached the point where technical control and the economy demanded by business are much better realized than in a smaller unit.

The Rockefeller Institute near one of the plants of the company has given valuable aid in the recognition and handling of animal diseases, and the essentials of this knowledge are available for all of the farms of the company. The nutritional problem is a fundamental one, "and we believe," said the speaker, "that with the various experiments and demonstrations we have a solid basis on which to work." In his later statements, Mr. Jeffers referred to the small farm and its relationships to the milk situation. He regretted that in general the agricultural college is not giving this matter the anticipated support, dealing, if the speaker was rightly understood, rather with the larger problems of the country.

Certain matters of administrative detail are essential. Mass production is a necessity in these days, yet work must be provided for the small farm that it may be successful. Mass production necessitates centralized control, but there should be decentralized operation. It is difficult to tell where one should end and the other begin but certain features have been worked out. In the growing of crops, for example, the centre decides on kinds of soil, fertilizer, seeds, general methods of procedure, and some items like these, but the actual work should be performed by decentralized units.

Experiments have been made by the Walker-Gordon people which have resulted in food stuffs at not more than half the cost under the older systems, and the boys on the farms, decentralized, are making more money. In breeding, the stock is centrally selected, the care and development of the young cattle being decentralized on a "pound-gain" basis. As to the manure, it is proposed to collect it as soon as produced, treat it chemically and make of it a dry substance. This avoids possible contamination of the milk, the nuisance of the manure pile, the health menace of the manure-bred house fly, and there is no economic loss through failure to use it at its maximum value time, for the dried fertilizer can be kept indefinitely and applied to the land when needed.

The "merry-go-round", explained by Mr. Jeffers during his second address, is a moving-sidewalk device with a speed of about fifteen feet per second. In one section the cow is given a thorough washing, in the next it is dried and further along there is the milking machine with automatic cleansing of utensils. A machine with space for fifty cows is nearing completion.

Throughout the discussion by Mr. Jeffers ran the

general thread, with occasional definite statements, that processing milk will not better the original product, and pasteurized milk will be no better than the original. For the benefit of those who think they must have pasteurized milk, the Walker-Gordon Farm pasteurizes a part of the product, but at the same time it holds that it is possible, with proper safeguards to furnish its customers with a safe, high standard, raw milk, the certified milk.

Dr. J. G. Hardenberg, veterinarian for the Walker-Gordon Laboratory, presented a scholarly discussion of the milk question, with emphasis on the possibilities of a high standard for certified milk.

## GENERAL MEETING OF THE INTERNATIONAL SOCIETY OF MEDICAL HYDROLOGY AND INTERNATIONAL LEAGUE FOR COMBATING RHEUMATISM

These two important societies will hold their joint meeting in the autumn of this year, Oct. 13-18, in Budapest, under the presidency of Baron Dr. Alexander Korányi. The scientific discussions are the following:

I. Hydrology: 1. The Action of Hypertonic Waters. 2. The Use of Waters and Baths in Cardio-Vascular Affections.

II. Rheumatism: 3. The Relation between Acute and Chronic Rheumatism in the various countries. 4. The Haematology and Serology of Rheumatism.

The scientific discussions will be completed by visiting the famous bathing institutions of Budapest, the City of Baths, and the most important health resorts of Hungary. All ordinary members of the society are entitled to join the General Meeting. Information about joining the International Society of Medical Hydrology may be had through the representative, Dr. Guy Hinsdale, White Sulphur Springs, West Virginia. Non-members, as guests, are to apply at the central bureau International Society of Medical Hydrology, 139, Marylebone Road, London W. I.

## SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

**September 27-28**—New England Surgical Society. Detailed notice appears on page 1227, issue of June 6.

**October 7-19**—New York Academy of Medicine. Detailed notice appears on page 468, issue of February 28.

**October 13-18**—General meeting of the International Society of Medical Hydrology and International League for Combating Rheumatism. Detailed notice appears above.

**November 4, 5, 6 and 7, 1929**—Clinical Congress and Eighth Annual Meeting of the American College of Physical Therapy. See page 149, issue of July 18, for details.

**December 2-6**—The Radiological Society of North America. Complete notice appears on page 149, issue of July 18.

**May 5-10, 1930**—First International Congress on Mental Hygiene. See page 191, issue of July 25, for details.

### DISTRICT MEDICAL SOCIETY

#### Norfolk District Medical Society

Schedule of meetings for the year 1929-1930. Invitations have been sent to the speakers and the list is true only on condition of their acceptance.

**October 29**—Medfield State Hospital, Harding, Massachusetts. Papers by the Staff.

**November 25**—Roxbury Masonic Temple. Dr. Roger Graves, "Urology from the Standpoint of the General Practitioner." Surgical discussion. Medical discussion.

**January 27, 1930**—Roxbury Masonic Temple. "Recent Advances in Diagnosis and Treatment of Syphilis." Speaker to be announced.

**February 24, 1930**—Roxbury Masonic Temple, 8:00. Dr. Edwin H. Place has been invited to read a paper and has been given carte blanche in the selection of his subject.

**March 24, 1930**—Roxbury Masonic Temple. Dr. Burton E. Hamilton. Subject to be announced.

**May 6, 1930**—Annual Meeting. Program to be announced.

The Censors meet for the examination of candidates, November 7, 1929, and May 1, 1930, in the Roxbury Masonic Temple, 171 Warren Street, Roxbury, at 4:00 P. M. Applications must be in the hands of the Secretary at least one week previous to date of examination.

Owing to inability to engage the Roxbury Masonic Apartments on the last Tuesday of the month, such meetings as will be held in the Roxbury Temple will be held Monday evenings as per the dates above.

FRANK S. CRUICKSHANK,
Secretary, Norfolk District Medical Society.

## BOOK REVIEWS

*The Technic of Local Anesthesia.* By ARTHUR E. HERTZLER. C. V. Mosby Company. Fourth Edition. 278 Pages. Price $6.00.

The Fourth Edition of Hertzler's book of 278 pages, 146 illustrations and a good index is thoroughly practical.

The opening chapter is devoted to the drugs employed, their preparation, their action, and applicability. The following chapter on technic of administration is replete with practical suggestions regarding needles and syringes, their care and sterilization, preparation of the patient, preliminary hypnotic, and methods of injection. Endermic infiltration, nerve blocking, perineural infiltration and edematization are then described. These pages indicate that the author prefers simplicity of apparatus, simple apparatus well cared for. The remainder of the volume is devoted to the regional application of various methods of local anesthesia. The neural anatomy is described and usually illustrated. The chapters concerning operations on the scalp, cranium, face, jaw, tongue, ear, mastoid, gasserian ganglion, trifacial nerve, tonsil and larynx describe in detail the avenues of approach as determined by work on the cadaver and give many useful practical suggestions regarding the application of these procedures in the living. The 34 illustrations in this section of the work are helpful amplifications of the text. In speaking of mastoid operations the author, contrary to Kulenkampff, finds no contraindication to the use of local anesthesia in sinus thrombosis and temporal abscess. He suggests a water bag as a more comfortable pillow than a sand bag or blanket.

A number of pages are devoted to operations on the thyroid. For a complete description of the author's technic, the reader is referred to the author's book on Diseases of the Thyroid Gland, C. V. Mosby Co.

Under operations on the spine the use of the author's laminectomy trephine is described. This consists of an ordinary 1¼ inch trephine from which 3/5 of the cutting edge has been removed. The nonsensitiveness of the posterior portions of the dura and cord is noted.

The use of local anesthesia in operations on the thorax is excellently presented. It is terse and practical.

In Chapter XI on Abdominal operations much attention is properly given to the nerves of the abdominal wall and the sensitiveness to crushing, stretching, and cutting of its various tissues, the parietal and visceral peritoneum and the contained organs. The chapter is concerned chiefly with the infiltration

use of local anesthesia. Although the use of spinal anesthesia is subsequently given, we are surprised that no mention of it is made in this chapter. We are pleased, however, to quote a few statements made here in regard to local infiltration.

"He should approach his patient prepared to use the method of anesthesia appropriate to the conditions and not predetermined to see whether the operation can be done under local anesthesia . . . If an organ which is ordinarily readily accessible is found when the abdomen is opened to be firmly bound down, general anesthesia should be resorted to at once before an attempt is made to loosen the adhesions. Such an attempt would have no other effect than to hurt the patient and destroy his equanimity. Similarly, if an abscess is adherent it can be easily drained; but if the free peritoneal cavity must be traversed, requiring packing off with sponges, general anesthesia is indicated. Introduction of a protective tamponade under local anesthesia should not be attempted. Nitrous-Oxide can be easily and quickly administered and serves perfectly in most cases. If a more protecting operation is required, or if greater laxity of the abdominal muscles is demanded, this can be secured by gas, the switching to ether in the hands of a skillful anesthetist is the matter of but a few minutes". (The above presupposes an approach under infiltration and does not refer to a possible choice of spinal anesthesia, as above noted.) Operations on the stomach, colon, appendix and gall bladder under local infiltration are then described. The author is apparently fond of simply draining the gall bladder under local infiltration "to tide the patient over critical periods until such time as a radical operation under ether can be safely undertaken." He concludes the chapter with the statement that "sometimes splanchnic anesthesia will solve the problem but at the present time it has added little".

Paravertebral anesthesia is theoretically an ideal procedure but the author believes that it has a very limited field of usefulness. The number of separate injections renders it a time consuming and trying method for the patient. Other methods of local anesthesia are preferable in all conditions except operations on the thoracic oesophagus. Thus in other thoracic operations he prefers to reach the intercostal nerves at the angle after the nerves have passed between the intercostal muscles. He prefers straight infiltration in grave gall bladder and kidney cases and spinal anesthesia in pelvic cases.

Splanchnic anesthesia by the posterior (Kappis) and anterior routes is described. The author is not enthusiastic about the posterior method on account of the "widely diffused" nature of the semilunar ganglia and because of the particular nature of the fat in which they are embedded which he believes acts as an insulator against the absorption of the anesthetic fluid. The anterior route necessitates the opening of the abdomen first. Adherence of viscera may preclude the approach to the ganglia. Although theoretically the method is ideal in eliminating shock, the author believes it is still to be classed as a "stunt" and that at present it "presents a hope rather than an achievement".

The author is convinced of the value of sacral blocking in perineal operations. When it has failed, more or less, he has aways supplemented local infiltration. By so doing he has never had to resort to general anesthesia. Failures, he believes, are due to gross errors in injecting the fluid and to the use of too small an amount. Two ounces of a 1% solution give more certain results than half this amount of twice the strength.

The use of spinal anesthesia is covered in about three pages of text.

There follow two practical chapters on operations for hernias, and separate chapters on operations on the male and female external genitals, about the anus and on the arm (including branchial plexus blocking) and on the leg.

The work appeals to the practical surgeon because the author, although an advocate of local anesthesia, denounces again and again the use of any of the various methods as a mere "stunt". He insists that there must be a definite indication. He has had a wide experience. He still uses general anesthesia. He is not a faddist. His uppermost concern is what is best and safest for the patient. Although the technic of the various methods of local anesthesia is excellently described and illustrated, his work is much more. He endeavors to indicate the selection of method for the special case. His evaluation of the advantages and shortcomings of the various procedures in actual practice is based upon operative experience and surgical judgment. This is the most valuable feature of the book.

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*A History of the Medical Department of the United States Army.* By COLONEL P. M. ASHBURN, Medical Corps, U. S. Army, Author of "The Elements of Military Hygiene." Houghton Mifflin Company. The Riverside Press, Cambridge, 1929.

Colonel Ashburn tells us in the first part of this most interesting history of the Medical Corps of the Army, that its beginnings were coincident with that of the Colonial Army at Boston in 1775. "Prior to that time there had been no army, merely a few colonial militia organizations, widely separated, without a common command, modeled upon the English regiments. . . . The surgeons of these organizations, when they had any, were appointed locally, usually by the colonels."

The book is divided into six parts, with a sketch of Surgeon General Merritte W. Ireland, by General Pershing, as a finale. Part one takes up the first century, from the Revolution to 1873, part two takes us to the Spanish-American War and the next section is entirely devoted to the medical department in this war. Next comes the period from our peace with Spain to the World War. Part five takes up the World War, and the conclusion of the volume brings the story of the Medical Corps to 1928. There is much of interest in the appendix, especially Dr. Leary's letter concerning Dr. Benjamin Church of the Revolution and Beaumont's account of the case of Alexis St. Martin.

At the time of the Revolution there were few medical men in the Colonies of European medical training, and the struggles of Morgan, Shippen, Rush and Stringer, to bring some order and efficiency to the young Medical Corps are graphically described. Scant and poor supplies, smallpox, dysentery, and pneumonia, made urgent problems, which had to be combatted as best they could. In the south, in the expedition against New Orleans, there was much sickness from scurvy, malaria, "intermittant, remittant, bilious and ardent fever." Liver abscess and yellow fever were common, so it

may be seen that the English were not exempt from the diseases incident to campaigns.

Turning to the Civil War, it is of interest to note that at that time our regular army consisted of but fifteen thousand officers and men. The medical officers "had neither experience nor training for anything larger than post practice and peace time administration, there were only 114 officers in the Department. There were no nurses except those detailed from the line." Surgeon Tripler reported to McClellan that he found the ambulances "In general use as pleasure carriages, for idlers, and accommodation cabs, for conveying officers and men." How Surgeon General Hammond brought the Medical Corps to some efficiency, against constant interference by stone walled bureaucracy and red-tape is well told. The horrors of the prison camps, north and south, are fittingly left untold.

The section on the Spanish War is one of the most interesting; the country was placid, the medical lessons of the Civil War were forgotten. Again the fault of the times, plus inertia, lack of knowledge of the science of medical sanitation and hygiene, brought havoc to our army at home and in Cuba especially. All this is described by Colonel Ashburn in a way that no one can forget.

With what pleasure and pride does the reader go on to the section of the World War; efficiency, coolness, good organization, nothing is wanting, and the story told of the Medical Corps in this section, in its work at home and abroad, gives a clear and illuminating picture that must be read in detail to be appreciated.

Colonel Ashburn and the Army Medical Corps are to be congratulated on this volume.

When will a similar story of the Medical Corps of the Navy be produced? Written by some Naval surgeon with patience to look into the past, as Colonel Ashburn has—it would prove equally of interest with the present volume.

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*Care of the Mouth and Teeth.* By HARVEY J. BURKHART, D.D.S., Director, Rochester Dental Dispensary, Rochester, N. Y. Cloth, 45 pages, New York, Funk & Wagnalls Company, 1928. Price 30c.

This booklet of the National Health Series, edited by the National Health Council, has been written with a distinctly conservative trend, notwithstanding in the first paragraph the author quotes Sir William Osler's broad statement that "Oral Hygiene is one of the outstanding public health matters, the promotion of which will do more to prevent disease and to promote the life of the human race than any other single activity in the whole field of modern sanitation."

Dental infection, and the damage it does to other structures of the body, is briefly discussed, with the recommendation that every source of infection about the mouth be promptly removed. That this may be done, under some conditions, without loss of teeth will interest those with a conservative attitude toward this important subject. Quite properly the author stresses the importance of the care of the mouths of children, beginning with the education of the expectant mother. Thought and attention to mouth hygiene of the infant and the growing child will be important factors in the normal development of the child as a whole. A large number of cases of juvenile delinquency, irritableness, and incorrigibility have been cured by removal of the cause so frequently found in the mouth.

Pyorrhea, that pest of middle age, also receives attention. That "there is no definite knowledge at the present time as to the cause of pyorrhea", and that "pyorrhea will rarely or never be found in a mouth where strict attention has been given to cleaning and care of the teeth", is open to challenge, but the common sense suggestions regarding the care of the mouth in adult life merit cordial support.

In the last chapter the author discusses the relation of diet to diseases of the teeth. Nutrition probably holds the key that will unlock the door admitting us to the secrets of the cause of degenerative changes in the teeth and supporting structures. Dr. Burkhart offers sample menus which will help build bone and teeth. Parents and those who have difficulties with teeth will profit by the perusal of this concise exposition of the subject.

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*Hermann M. Biggs, M.D., D.Sc., LL.D., Physician and Statesman of the Public Health.* By C.-E. A. WINSLOW, DR.P.H. Lea & Febiger, Philadelphia, 1929. Price $5.00.

There is a foreword by Dr. William H. Welch. In the four hundred pages which follow Professor Winslow presents in an entertaining way carefully accumulated information to make us better acquainted with a man whose personality tended to be effaced by his work.

The book is, however, much more than a biography. Dr. Biggs's professional career was interwoven with the development of what has come to be regarded as the proper method of dealing with important public health problems, including that of tuberculosis. We now look upon the promotion of personal health as a function of government. The acceptance of this idea has been gradual. Dr. Biggs was prominently identified with its development. Progress was made from time to time by taking advantage of interesting political situations which Professor Winslow is able personally to recall. It was on such occasions that Dr. Biggs displayed the peculiar talent which doubtless has suggested the appellation "Statesman of the Public Health".